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### LYMPHADENOMA.<sup>1</sup>

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It has been claimed that of the seven cases described by Hodgkin in his famous paper, "On Some Morbid Appearances of the Absorbent Glands and Spleen", only one, or possibly two, would now be classified as Hodgkin's disease. Even in this year of grace, with the assistance of the histologist and the hæmatologist, it may be said that "diagnosis is difficult and judgement fallacious". The histological findings are often difficult of interpretation, and depart very widely from the classical description of Reed and others, whilst blood examinations are often of more value in excluding the diagnosis than in confirming it. Nevertheless, amongst the ill-defined group of progressive enlarge-

ment of the lymph glands there are certain cases concerning which the clinician, the pathologist and the hæmatologist can reach ultimate unanimity, even if this happy issue is reached only over the autopsy table.

I do not propose to fatigue you with a laboured account of the symptomatology of Hodgkin's disease. The sorry tale is well known to you—a stage of glandular enlargement, frequently not quite so free of pain and tenderness as many textbooks state, and a terminal cachexia with fever of an irregular, or quite commonly a relapsing, type, secondary anæmia and in many instances the added horrors of mechanical obstruction to the airways and the blood vessels by huge masses of enlarged glands.

In all cases biopsy is essential to confirm the clinical findings, and when this precaution has been omitted the diagnosis must be considered more or less suspect. On clinical grounds it is sometimes impossible to differentiate between tuberculous glands, especially if there is no evidence of caseation, some varieties of leuchæmia in the aleuchæmic

<sup>1</sup> Read at a meeting of the New South Wales Branch of the British Medical Association on November 29, 1934.

phase, lymphosarcoma and reticulum cell sarcoma, rare instances of glandular metastasis from cryptogenic new growth, occasional cases of profound simple hyperplasia and Hodgkin's disease itself. Thus any help from the morbid histologist and hematologist is not only welcome, but necessary.

In the ten years ending December 31, 1932, the records of the Sydney Hospital reveal that 69 cases were ultimately diagnosed as lymphadenoma. These figures do not represent the total number of patients who were admitted with a provisional diagnosis of lymphadenoma, which investigation subsequently proved to be inaccurate. Moreover, on careful scrutiny it appears that of these 69, at least 13 must be discarded as examples of glandular tuberculosis, lymphosarcoma and reticulum cell sarcoma, leucæmia of the aleucæmic variety, cases of secondary neoplastic invasion, and certain obscure glandular hyperplasia. In three cases the history and the general information provided were insufficient for diagnosis, but probably sufficient to exclude the likelihood of lymphadenoma. In fact, the eventual diagnosis affixed seemed, in some cases, to depend upon the diagnostic vagaries of the house physician concerned, rather than on a just appraisal of the clinical and pathological findings.

Biopsies were performed on 41 patients; of the remainder, some presented no accessible glands, others were too ill for any form of interference, and in a few cases the patient would not consent. In 22 cases the pathological report either confirmed or acquiesced very guardedly in the clinical diagnosis, in six the finding was controverted, and in the remainder the report was inconclusive.

Of the 53 cases in which a diagnosis of lymphadenoma was sustained by clinical evidences and/or biopsy or autopsy, certain interesting facts emerge. The glandular enlargement commenced in the neck in 34, in the axilla in two, in the groin in four, in the mediastinum in two and in the abdominal glands in four. In the remaining seven cases the site of onset is not mentioned. Of those patients whose condition originated in the cervical lymph nodes, a considerable number gave a history of an antecedent tonsillitis; one gave a history of mumps and one of injury to the chin. Of the four patients whose disease apparently originated in the inguinal region, two gave a history of antecedent injury to the leg on the affected side, whilst the third had been operated upon for appendicitis a month or so before.

The incidence according to age groups is as follows: One to ten years, eight; ten to twenty years, 16; twenty to thirty years, 17; thirty to forty years, seven; forty to fifty years, five. There were 44 males and nine females. These figures, however, are apt to be misleading, inasmuch as the hospital provides proportionately more accommodation for males than for women or children. The youngest patient in this series was a child of three years, the eldest a man of fifty.

The duration of the disease varied from seventeen weeks to six and a half years, and in a number of the more chronic cases there was a history of remissions during which the patient felt comparatively well. Nevertheless, in all those cases which could be traced the outcome was fatal. The only constant blood change was the presence of a progressive secondary anæmia in the later stages of the disease. The white cell counts varied greatly. Early cases tended to show a moderate increase in the polymorphonuclear elements, but in the later stages the findings ranged from a leucopenia to a moderate leucocytosis. Persistent lymphocytosis, when present, strongly suggested an aleucæmic leucæmia.

One patient in the later stage of the disease presented a leucocytosis of 13,000, with 31.5% of eosinophile cells. The total count in another was 800, of which 86% were neutrophile cells.

Autopsy revealed several cases in which there was gross invasion of surrounding organs; in one a lung was almost completely destroyed by tumour growth. This has been classified as Hodgkin's endothelial sarcoma, but serves to illustrate the impossibility of drawing any sharp line between the diseases of the lymphadenoma group.

H. M. Gordon observes that: "Lymphadenoma occupies a position in the marches of medicine forming a link between the granulomata of known etiology and malignant disease." In this series, as in others, the large number of cases in which the cervical glands were first to enlarge strengthens the theory of an infection primarily tonsillar or buccal, but not infrequently bronchial or alimentary in origin. It has been previously observed, as in some of these cases, that when the axillary, femoral or inguinal glands are primarily affected, there has been a history of injury and infection in the corresponding part; but it must be conceded that one could present a succession of cases ranging from a picture of acute infection to one of pure neoplasm, with infinite gradations between these extremes. Some hold that the disease is a manifestation of tuberculosis, more particularly of the avian type. L'Esperance asserted that infection of lymphadenomatous tissue produced tuberculosis in chickens. Utz and Keatinge, in this country, produced evidence in support, but Van Rooyen, Garrod, Stewart, Wallhauser and Steiner have failed to confirm their findings.

The weight of evidence seems, therefore, strongly opposed to the tuberculous origin of this disorder, but quite recently an interesting observation was made by Steiner. Proceeding from the knowledge that almost all adults have been sensitized to large doses of tuberculin, he tested 35 patients suffering from lymphadenoma with progressive doses of human and avian tuberculin given intracutaneously. He found that there was more or less complete absence of sensitization to both avian and human tuberculin protein in the cases of lymphadenoma. He concluded that either (1) the process of lymphadenoma desensitizes its victims to these tuberculin

proteins, or (ii) that lymphadenoma usually occurs in persons in whom development of the normal sensitization to the tuberculin protein is impossible. His final inference is that it is difficult to conceive either of these phenomena as occurring in a disease absolutely unrelated to tuberculosis.

The Rose research workers under H. M. Gordon successively exculpated spirochaetes, human, bovine and avian tubercle bacilli, and lastly various yeasts. Gordon, however, repeatedly succeeded in producing encephalitis in rabbits by intracerebral inoculation of lymphadenomatous tissue. The encephalitis produced runs true to type, and has been frequently used as a confirmatory diagnostic measure in doubtful cases. Van Rooyen obtained 15 positive results out of 20 cases of lymphadenoma, and negative results with glands in other diseases. Gordon also found bodies, which he called Hodgkin bodies, in fluid aspirated from lymphadenomatous glands during life, and demonstrated their similarity to the so-called Paschen bodies, which have been proved to be the causal agent of vaccinia.

However, Friedeman and Ekeles showed that an agent could be derived from normal human marrow and spleen which produced an encephalitis, apparently identical with that produced by lymphadenomatous tissue. They further proceeded to show that the agent was present in human leucocytes, and that it was probably not a living virus, but a toxin indistinguishable from the proteolytic ferment which Jockmann found in normal human leucocytes. Whether these conclusions are correct or not, it seems that it is possible to distinguish lymphadenoma from other forms of glandular enlargement by the presence in lymphadenomatous tissue of an encephalitogenic agent which is absent in other varieties of glandular disease.

A third group of observers claim that lymphadenoma is neoplastic in origin, and choose to link it up with the myeloses and lymphadenoses (myeloid and lymphatic leuchæmias), and assign it to a category known as the reticuloses, of which it forms only one of a number of disorders of reticular tissue.

#### Conclusion.

In conclusion I may point out that:

1. Lymphadenoma is one of a group of diseases which intervene between the known granulomatoses and malignant disease.

2. Its ætiology is unknown; its relationship to tuberculous, spirochaetal, mycotic and viral infections has not been established.

3. Diagnosis depends upon clinical evidence, biopsy reports and blood examinations. A persistent lymphocytosis should suggest an aleuchæmic lymphatic leuchæmia.

4. To these methods of diagnosis the intracerebral inoculation of rabbits should now be added.

#### HODGKIN'S DISEASE.<sup>1</sup>

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THIS paper is a review of Hodgkin's disease as found in the biopsy and autopsy material of the Royal Prince Alfred Hospital during the last ten years. In this period there were 2,473 autopsies, and in this number, eight patients, or 0.32%, were found to have died of Hodgkin's disease. In the biopsy material twelve cases of the disease were encountered. Three of these came to autopsy in the hospital, so that the biopsy material provided only nine cases in addition to those first mentioned.

As might be expected, the proportion of patients dying of Hodgkin's disease varies in different hospitals. In a series of 7,253 autopsies reviewed by Barron,<sup>(1)</sup> 24 patients, or 0.32%, died of the disease, while among Symmers's<sup>(2)</sup> series of 8,485 autopsies, 14 cases were found, an incidence of 0.16%.

Of the 17 patients under consideration, 15 were males and two females, a ratio of 7.5 to 1. All of the eight patients who came to autopsy in the hospital were males. Wallhauser,<sup>(3)</sup> in a series of 1,447 cases collected from the literature, found that the ratio was 2.3 to 1.

The cases of the present small series are classified according to age in Table I. The autopsy and biopsy cases are separated, since the figures given for the former group refer to the age at death, while those of the latter group refer to the age of the patients at the time the biopsy was taken. For comparison the larger series of Longcope,<sup>(4)</sup> Barron,<sup>(1)</sup> and MacNalty<sup>(5)</sup> are given.

TABLE I.  
*Cases of Hodgkin's disease classified according to age.*

Decade.	Cases occurring at the Royal Prince Alfred Hospital during the ten years ended June 30, 1934.		Series of cases recorded by:		
	Autopsy Cases.	Biopsy Cases.	Longcope. <sup>(4)</sup>	Barron. <sup>(1)</sup>	MacNalty. <sup>(5)</sup>
1st	2	1	22	—	6
2nd	—	—	31	2	7
3rd	2	1	29	5	13
4th	1	1	35	7	7
5th	2	3	16	2	4
6th	1	3	9	6	2
7th	—	—	4	2	—
8th	—	—	—	—	—
9th	—	—	1	—	—
Age not given	—	—	3	—	—
Totals ..	8	9	150	24	39
Average age	30.6 years	39.5 years	—	—	—

The finding of Barron that 50% of the cases occurred between the ages of 21 and 40 years is

<sup>1</sup> Read at a meeting of the New South Wales Branch of the British Medical Association on November 29, 1934.

true also of MacNalty's series, but not of that reported by Longcope nor of the small group of cases under consideration. Wallhauser noted that very few cases occur at the age of puberty; "of 33 cases in children 15 years old or younger, only two occurred between the ages of 12 and 14".

It will be convenient to commence with the typical histological picture of Hodgkin's disease as seen in the lymph glands, but first the normal histology of these structures should be considered.

A normal lymph gland is seen to consist of a cortex and a medulla and to be surrounded by a fibrous capsule which sends trabeculae into the substance of the gland. In the cortex there are numerous lymph follicles which contain a pale staining germinal centre around which there is a condensation of lymphocytes. Lymph sinuses separate the cortex from the capsule and from the trabeculae. In the medulla the arrangement is less regular and here the tissue is broken up into reticulated lymphoid cords, the cords being separated from each other by the lymph sinuses of the medulla. In both the cortex and medulla the lymphocytes, which form the bulk of the gland, are supported by a very delicate reticular stroma. This stroma consists of two elements: (i) the reticular fibres and (ii) the reticular cells.

The reticular fibres, by dividing and anastomosing, form a network which extends throughout the whole gland. The fibres are not seen in the normal routine histological preparations, but may be impregnated with silver and hence are termed argyrophile fibres.

The reticular cells also form a sponge-like network and are closely applied to the reticular fibres. They are found in various stages of differentiation. The most primitive are the cells of the undifferentiated syncytial reticulum. These have pale-staining vesicular nuclei and irregularly stellate cytoplasm, the processes of which are continuous with those of adjacent cells of similar nature. In animals vitally stained with such pigments as lithium carmine these cells take up almost no stain. According to Maximow,<sup>(6)</sup> whose description of the histology of the lymph gland is being followed here, these undifferentiated syncytial cells are of a very similar nature to the cells of the primitive mesenchyme, and from them most of the other cellular elements of the gland are derived. Developed from these cells, but somewhat more differentiated, are the "reticular cells proper", or histiocytes. They take part in the formation of the cellular reticulum, but are larger than their parent cell and have larger pale vesicular nuclei. Unlike them, they can ingest large amounts of lithium carmine in vitally stained animals.

The reticular cells are not very easily seen in a normal lymph gland, but their pale nuclei are found among the densely packed lymphocytes. Reticular cells proper form the lining of lymph sinuses, and in certain pathological conditions, such as reactive hyperplasia, these cells become conspicuous and form a network which incompletely fills the sinuses.

Macrophages are developed from the reticular cells proper. They are not attached to the fibres of the reticulum, but are free cells. In addition to being able to ingest lithium carmine, they can ingest cellular *débris*. These cells may become very numerous in certain inflammatory and reactive conditions and are often seen in large numbers in the lymph sinuses.

The lymphocytes in the gland are small, medium sized and large. The large lymphocytes are found mainly in the germinal centres of the lymph follicles, but also occur in other parts of the gland. They are thought to be developed from the cells of the undifferentiated syncytial reticulum and to give rise to medium sized lymphocytes which in turn give rise to the small lymphocytes which form the bulk of the lymph gland parenchyma.

#### Histological Pictures of the Lymph Glands.

Some characteristic histological pictures of the lymph glands in Hodgkin's disease may now be described.

In a gland removed surgically from the neck of a female patient, aged fifty-six years (J.1309), the changes, though showing definite evidence of Hodgkin's disease, are comparatively early. This gland has lost its normal architecture: there is no division into cortex and medulla, and no lymph follicles can be recognized. Lymphocytes, though still very numerous, are much reduced in number. There has been a diffuse overgrowth of reticulum cells, which are enlarged and which have abundant pale-staining but somewhat ill-defined cytoplasm. They are not clearly separated from adjacent reticular cells. Most of them have pale vesicular oval nuclei with well defined but not very deeply stained nuclear membranes; the chromatin network is delicate and a nucleolus may or may not be seen. Here and there are occasional nuclei which, though still pale-staining, are much larger than the others, and there are some which have large and dark-staining nucleoli (see Figure 1).

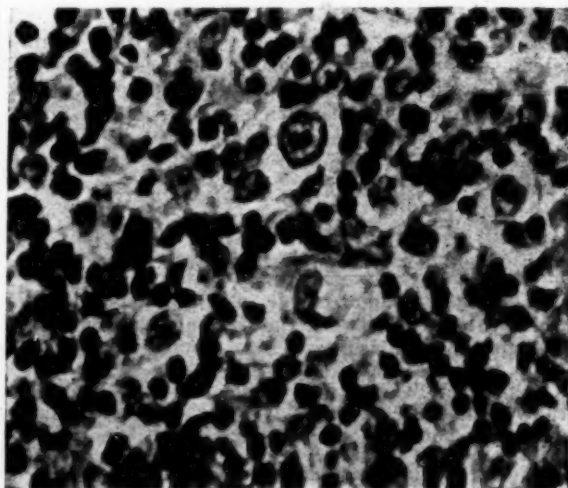


FIGURE 1.

An early stage in lymphadenoma. Lymphocytes relatively reduced in number. Reticulum cells hyperplastic, several with large hyperchromatic nucleoli and one with a deeply stained nuclear membrane. From a female patient, aged fifty-six years. Biopsy number J.1309. Iron hematoxylin and van Gieson's stain.  $\times 550$ .

The changes described so far would not be inconsistent with some form of simple hyperplasia of the cellular reticulum, but here and there there is evidence that a further development in the cells is taking place.

Occasional cells are found in which the nucleus, as well as being enlarged, is hyperchromatic. There is a fairly heavy nuclear membrane and a large deeply stained nucleolus. The cytoplasm has become slightly better defined and the cell shows a tendency to be isolated from those surrounding it.

The next type of cell to be described is one which is very characteristic of Hodgkin's disease and is the so-called "Sternburg cell", "Dorothy Reed cell" or "lymphadenoma cell".

In the gland being described these are not very numerous. They are small giant cells which are almost spherical in shape, and appear to be well defined, since they usually lie in vacuoles in the tissues and have lost their fine protoplasmic connexions with neighbouring cells. The cytoplasm may be only slightly more deeply stained than that of other reticular cells, but there are two, three, four or more nuclei which are definitely hyperchromatic. Each nucleus has a deeply stained nuclear membrane and a prominent nucleolus, the remainder of the chromatin being sometimes very scanty and sometimes fairly abundant. It is very usual to find a lymphadenoma cell with two nuclei which partially overlap each other (Figure II).

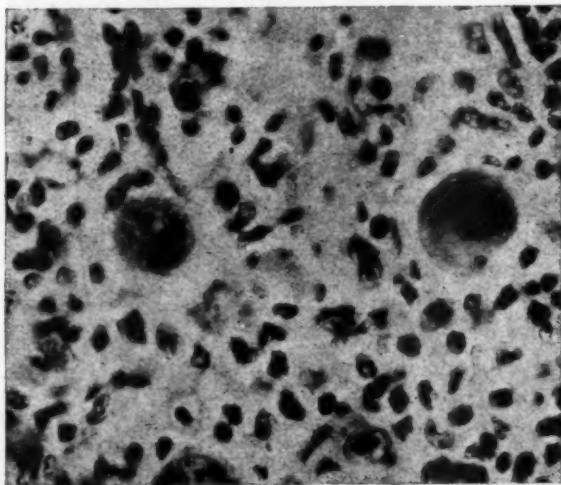


FIGURE II.

Two large multinucleated lymphadenoma cells (Dorothy Reed cells). These have well defined cytoplasm, which is not connected with that of adjacent cells. Nuclei overlap each other at their edges and have deeply stained nucleoli. Other cells include reticulum cells and also lymphocytes, which are reduced in number. From a male patient, aged forty-one years. Biopsy number N.753. Hematoxylin and eosin stain.  $\times 650$ .

Another characteristic picture is produced when, as pointed out by Pullinger,<sup>(7)</sup> two nuclei are in close contact and form a mirror image of each other.

There seems little doubt that these lymphadenoma cells are developed locally in the gland and that they are derived from reticulum cells. All gradations between the reticulum cell and the lymphadenoma cell can be found. Andrewes,<sup>(8)</sup> Reed<sup>(9)</sup> and most of the more recent authors hold this view. Symmers, on the other hand, considers that they

are derived from the bone marrow and are carried to the gland by the blood stream. He bases his opinion on the fact that lymphadenoma cells may be found lying free in capillaries of the lymph glands, and considers that the cells are identical with the megakaryocytes of the marrow. More recently Medlar<sup>(10)</sup> expressed a similar view.

In another part of the same gland the reticulum cells have become elongated and with the routine stains resemble fibroblasts. Van Gieson's stain gives the ground substance a faint pink colour which suggests that early fibrosis is taking place and that collagen is being formed.

The scarcity of fibrosis in the lymphadenoma tissue in this gland may perhaps cast some doubt on the diagnosis. Pullinger considers that in the absence of fibrosis no lesion can be considered as quite typical.

The present section has not been stained to show reticulum fibres, but Andrewes, Longcope and Pullinger have shown that an increase of reticulum fibres is the rule in these early lesions. According to Pullinger this increase precedes the visible fibrosis.

In a gland from another case (Biopsy Number F.45) the morbid process has reached about the same stage of development as that in the gland just described. Here, however, the staining is suitable to demonstrate the numerous eosinophile leucocytes which are present. Some of these are polymorphonuclear and are in every way similar to the normal eosinophile leucocytes of the blood. In others the nuclei are pyknotic and at first sight appear to be ovoid or spherical. Careful focusing shows that the nuclei are usually bilobed and that one lobe is often placed at a lower level than the other. In some eosinophile cells the pyknotic nucleus is not lobed and in others the small single nucleus is slightly vesicular.

Cells of this type are illustrated in Andrewes's early paper and they are referred to by Pullinger as eosinophile micromyelocytes.

The presence of eosinophile leucocytes has long been recognized as a characteristic feature of the histological picture of Hodgkin's disease. Reed, as well as Andrewes, described them in 1902. Andrewes found them in small numbers in normal lymph glands and considered that they were always demonstrable in Hodgkin's disease. They were present in great numbers in all but two of Reed's cases. Concerning their origin opinion is divided. Many authors, including Reed, Simmonds<sup>(11)</sup> and Boyd,<sup>(12)</sup> consider that they are derived from the blood. Ewing<sup>(13)</sup> traces their origin to extravasations of blood in the early stages of the morbid process. Other authorities, including Pullinger, consider that they are developed locally in the gland. Pullinger figures all stages in their development from reticulum cells. Reed holds that while the absence of a general blood eosinophilia might suggest a local origin, the cells are probably attracted to the lesion by some chemiotactic substance.

Plasma cells are moderately numerous in a few glands from this series. In some cases we found, as Simmonds did, that they were most numerous in the peripheral portions of the gland.

In a gland from another case (Biopsy Number H.583) a further stage of the morbid process may be studied. Here lymphocytes are hard to find and the whole gland is occupied by a very cellular fibrous tissue. The majority

of the cells still have vesicular nuclei and resemble fibroblasts, but in others the nuclei have become pyknotic and elongated so that they resemble the lamellar cells of the fibrous tissue. Lymphadenoma cells are a conspicuous feature, but they are more numerous in some areas than in others. Their nuclei are very hyperchromatic, so that the characteristic nuclear structure is not always seen.

This gland contains an area of necrosis, a change which is frequent in the present series. In the necrotic area the outlines of the original tissue may still be recognized, and in this respect the necrosis differs from the caseous necrosis of tuberculosis. Polymorphonuclear leucocytes, or their remains, are found in the necrotic tissue and are often very numerous (Figure III).

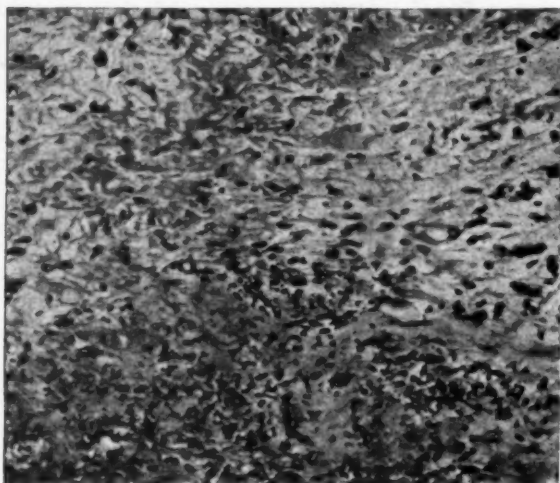


FIGURE III.

An area of necrosis, in which selective staining is lost, is seen in the upper part of the figure. In the lower part similar necrotic tissue is infiltrated by polymorphonuclear leucocytes. From a male patient, aged twenty-two years. Biopsy number H.583. Hæmatoxylin and eosin stain.  $\times 130$ .

The association of polymorphonuclear leucocytic infiltration with necrosis in lymphadenoma tissue has been observed by many authors, including Reed, Turnbull<sup>(14)</sup> and Pullinger, while Longcope noted that the change may be extensive after radiation therapy.

In many glands the fibrous element is more abundant but less cellular.

In one from an autopsy case (Case IV, gland from the neck) the whole gland shows a great reduction in the number of cells present. The fibrous tissue is in the form of broad sheets of hyaline material which contains so many vacuoles of various sizes that it resembles a sponge. Many vacuoles contain hyperchromatic cells, which are sometimes multinucleated. Other vacuoles are empty, suggesting that cells in them have fallen out or degenerated.

In a gland from another autopsy case (Case III) the hyaline change has progressed still further (Figure V). In both these cases there are more cellular areas in which the characteristic polymorphocellular picture of the disease is seen, but sometimes the whole gland is almost completely hyaline, so that it may be difficult to make a diagnosis.

It frequently happens that glands which are the seat of very advanced and fibrotic lesions contain areas in which the changes are very early.

One such gland was removed from the neck of a man aged forty years (Biopsy Number G.581). It is formed mainly of fibrous tissue which is almost completely hyaline. Embedded in this there are numerous islands

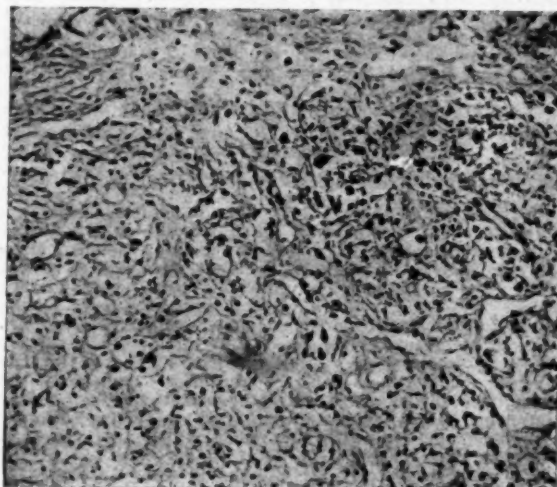


FIGURE IV.

Fibrosis in a lymphadenomatous lymph gland. The fibrous tissue forms a delicate network in the meshes of which are hyperchromatic reticulum cells and lymphadenoma cells. From Case IV, male, aged forty-four years. Hæmatoxylin and eosin stain.  $\times 130$ .

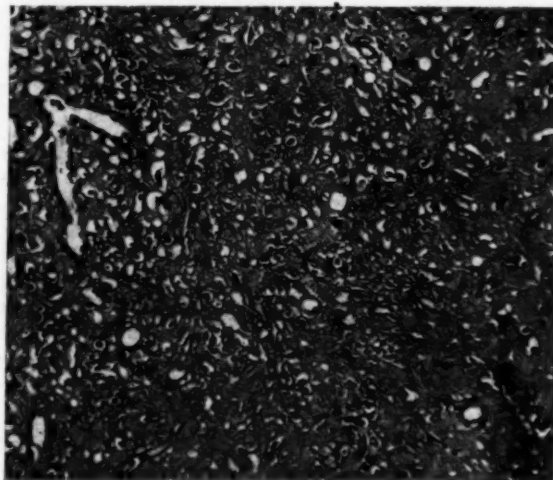


FIGURE V.

Much more dense fibrous tissue, which is almost hyaline. Lymphadenoma cells and reticulum cells lie in vacuoles. From Case III, male, aged five years. Hæmatoxylin and eosin stain.  $\times 130$ .

of lymphoid tissue with pale hyperplastic reticulum cells (Figure VI). Characteristic lymphadenoma cells are also present, as well as eosinophile and neutrophile leucocytes. Similar leucocytes, as well as many plasma cells, are scattered through the hyaline fibrous tissue.

It has been suggested by Pullinger that the dense and relatively acellular tissue can become revascularized and "from the adventitia of the ingrowing capillaries a new reticulum proliferates

and starts the whole process of lymphadenoma in the same area again".

So far the capsule has not been mentioned. In some cases it is only slightly thickened and in others it is infiltrated by lymphocytes. Sometimes the lymphadenoma tissue penetrates the capsule and infiltrates the surrounding fat.

In a gland from the series of autopsy cases (Case VI) the line of the original marginal lymph sinus can be seen. Inside this the adenoid tissue is replaced by lymphadenoma tissue which is rich in hyperchromatic reticulum cells and fibrosis. Outside it there are many similar cells, but here lymphocytes are more numerous. The lymphadenoma tissue outside the gland is bounded by a newly formed capsule of fibrous tissue—a process which has been described by Gibbons.<sup>(10)</sup>

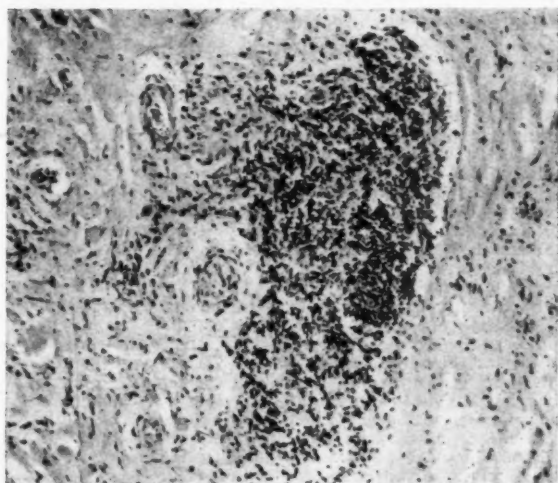


FIGURE VI.

A section from a gland which contains a large amount of very dense fibrous tissue. In the centre there is a cellular zone formed of lymphocytes and reticulum cells with occasional lymphadenoma cells, suggesting that a second active phase is commencing in the gland. There were many such cellular foci. From a male patient, aged forty years. Biopsy number G.581. Haematoxylin and eosin stain.  $\times 100$ .

#### Distribution of the Lesions in Hodgkin's Disease.

The appearances of the groups of enlarged glands of Hodgkin's disease and the firm, enlarged spleen with multiple suet-like areas are too well known to need further description.

Symmers classifies his cases according to the group of glands or the organ which showed the most pronounced involvement. In his series of fourteen cases he found the predominant involvement in the abdominal lymph glands in 28%, in the abdominal and thoracic nodes together in 43%, and in the cervical nodes in 7% of his cases. In other cases part of what he termed the "auxiliary lymphoid system" of the spleen, thymus or liver showed the most marked involvement. He found that involvement of the axillary lymph glands was sequential to involvement of the cervical or thoracic lymph glands and, similarly, appearance of the disease in the inguinal lymph glands followed that in the abdominal nodes. Symmers also noted that the submucosal lymphoid tissue rarely became affected by the disease.

#### Lymph Glands.

Notes on autopsies are too frequently silent as to the site of maximum glandular involvement, pathologists usually contenting themselves with a bare recital of the glands and organs involved.

In one of our autopsy cases (Case III) the mediastinal and bronchial glands were almost completely hyaline. In an anterior mediastinal gland and also in one from the base of the skull there was advanced fibrosis, but not so much hyaline change, while an iliac gland contained only a small amount of fibrosis and an inguinal gland showed the very earliest changes.

In the small series of autopsies under consideration the glandular manifestations were distributed as shown in Table II.

TABLE II.

Site of Affected Glands.	Number of Cases in which Glands were Involved.	Percentage.
Neck .. .. .	7	87.5
Axillae .. .. .	3	37.5
Mediastinum .. .. .	3	37.5
Abdomen .. .. .	8	100.0
Groin .. .. .	6	75.0

Glands affected by the disease were found in the abdomen in every case. In seven of the eight cases those round the pancreas were specially mentioned as being involved; in three cases those in the hepato-duodenal ligament or the *porta hepatis* were also affected.

#### Spleen.

The spleen showed the characteristic histological features of the disease in seven of the eight cases of the present series (87.5%). Enlargement was observed at autopsy in all seven. In two (Cases III and VI) the spleen was described as "very much enlarged", in four others it was "moderately enlarged", two of them (Cases IV and V) weighing 450 grammes and 540 grammes respectively. The enlargement was noted clinically in five of the cases. The typical "suet nodules" were seen in all of the seven cases, but in one (Case V) they were in the form of very small flecks. Necrosis in the lymphadenoma tissue was seen in all cases which showed the characteristic changes, but it varied in amount. In two cases (Cases IV and VI) it was widespread, but in one of these (Case IV) it appeared to be partly due to a large infarct. In two more it was moderate in amount and in two it was slight. Polymorphonuclear leucocytic infiltration in the necrotic areas was moderate in amount in four cases (Cases I, V, VI and VIII), slight in one (Case II) and absent in the remaining two cases. Fibrosis in the lymphadenoma tissue was present in six of the seven positive cases, being marked in three (Cases II, III and IV), moderate in two (Cases V and VIII) and slight in one case (Case VI).

Wallhauser, in reviewing the literature, found that there was splenomegaly in 226 out of 321 cases, an incidence of 70%. In Symmers's series of 14 cases the spleen was involved in eleven (78.5%),

the average weight of the organ being 802 grammes. In eight of these cases and in two others the spleen, while not enlarged, showed the typical histological features of the disease. In Turnbull's 37 cases, which were cited by Rolleston,<sup>(16)</sup> the spleen was involved in every instance.

#### *Liver.*

The liver was affected by the disease in seven out of eight cases, and in five of these it was found to be enlarged before death. In the seven livers mentioned a few small white areas of lymphadenoma tissue were seen near the surface in three instances (Cases II, IV and V). In one other case (Case VIII) it was seen to be surrounding the bile ducts. In two others (Cases VI and VII) the lymphadenoma tissue occurred in large tumour-like masses, those in one of them (Case VI) being related to large branches of the portal and hepatic veins.

Microscopically the lymphadenoma tissue was found in the portal canals in six cases (Cases I, II, IV, V, VI and VIII). The canals had become enlarged and the lymphadenoma tissue had encroached on the adjacent liver lobules. In one of these cases (Case V) there was only one area of definite lymphadenoma in the section, and this had apparently commenced in a portal canal. Adjacent canals contained an excess of lymphocytes, while one contained in addition some large reticulum cells with irregular hyperchromatic nuclei.

In the seventh case (Case VII) the lymphadenoma tissue was found in a large tumour-like mass which had compressed the adjacent liver tissue with consequent atrophy of the epithelial cells. Portal canals in this case contained no lymphadenoma tissue or lymphocytes, but very numerous polymorphonuclear leucocytes were present in these zones.

Necrosis of the lymphadenoma tissue had occurred in three of the seven cases (Cases IV, VI and VIII).

The Kupffer cells in four of the livers (Cases IV, V, VI and VII) were found to be hyperplastic, and many of them were loaded with blood pigment.

The lymphoid infiltration of the portal canals, mentioned in the above summary, was marked in one case (Case V) only, but in most of the others the morbid process had progressed so far that all canals exhibited advanced lesions. The lymphoid infiltration is of interest in that it lends support to the view that a preliminary lymphoid hyperplasia is the first change in Hodgkin's disease. Symmers lays particular stress on this early change and is of the opinion that lymphoid tissue occurs normally in the walls of the portal veins. It seems more probable that the precursors of lymphocytes occur in the portal canals and that these precursors are capable of differentiating in the first instance to form lymphocytes and later to form the hyperplastic reticulum cells and lymphadenoma cells of Hodgkin's disease.

Wallhauser found that the liver was involved in the disease in about 50% of cases reported in the literature.

In Symmers's fourteen cases the liver contained macroscopic nodules of lymphadenomatous tissue in eight and microscopic evidence of the disease in ten (71%). He described the preliminary lymphoid hyperplasia in the portal canals and the subsequent appearance of the characteristic tissue.

Jaundice was present in four of the eight cases under discussion. Barron considers that peribiliary infiltration in the liver is the most usual cause of this symptom, but pressure on large bile ducts by enlarged glands outside the liver is thought by other authorities to be a cause of jaundice in some cases.

#### *Lungs.*

The lungs were affected by the disease in three of the eight cases of the present series (Cases V, VI and VII).

In Case VI the lungs were large, pale and emphysematous, but were comparatively light in weight (right lung, 530 grammes; left lung, 440 grammes). A fairly coarse nodularity was felt throughout both lungs, the nodules being about the size of a split pea, almost white and ill-defined. No definite relationship to bronchi or blood vessels was noted on macroscopic examination.

Histologically the nodules are seen to vary in size, the smallest being about the size of a pulmonary alveolus and the largest being 4.5 millimetres in diameter and necrotic in the centre.

Comparatively early changes are seen at the periphery of the larger nodules. Here alveolar walls are thickened and contain large endothelial cells with pale-staining cytoplasm and oval vesicular nuclei. Occasional cells have irregular nuclei and some are hyperchromatic. Very few lymphocytes are found, as well as an occasional polymorphonuclear leucocyte. Capillaries are slightly dilated. Alveoli with walls as described often contain fibrin in which there are a few endothelial cells similar to those found in the alveolar walls (Figure VII).

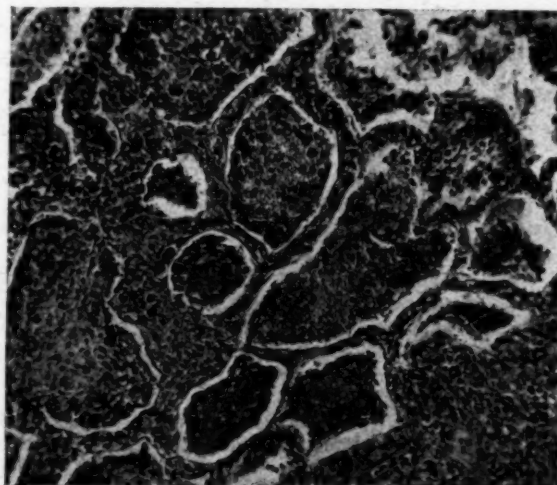


FIGURE VII.

Lymphadenoma tissue in the alveolar walls and alveoli of the lung. From Case VI, male, aged forty-nine years. Haematoxylin and eosin stain.  $\times 100$ .

In a part where the change is a little more advanced, alveolar walls are much thicker and contain a greater variety of endothelial cells. Many with pale vesicular nuclei are still present, but those with hyperchromatic and

very irregular nuclei are much more numerous than before. Occasional multinucleated lymphadenoma cells are seen, but these are not as well isolated as in a typical lymph glandular lesion. The plug of fibrin in the alveolus is now much more cellular, and the cells here, as in the alveolar walls, occur in great variety and include lymphadenoma cells. The cellular plug may appear to lie free in the alveolus, but often is in contact with the thickened alveolar wall in one or more places. In the centre of the nodule all remains of the original structure of the lung are lost. The lymphadenomatous tissue in the alveolar walls has fused with that in the alveoli and there is a widespread necrosis in many cases.

The change appears to have commenced in the adventitia of arterioles or venules, the majority of which are surrounded by a cuff of lymphadenomatous tissue. The media has often been reduced to a very thin layer of muscle fibres, but has not been completely destroyed at any one point in any of the vessels in the section. The intima in many cases is very much thickened by lymphadenomatous tissue which is present between the media and the very thin but intact endothelium. The blood is not obviously clotted in the lumina, but some very delicate shreds of fibrin may be present (Figure VIII).

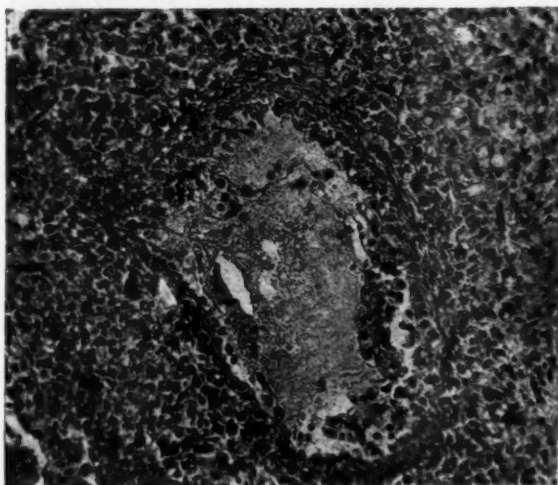


FIGURE VIII.

Lymphadenoma tissue in the lung. Transverse section of an arteriole showing lymphadenoma tissue in the adventitia, the outer part of the media and in the intima. The endothelial lining of the vessel has been partly detached in the upper half of the section. From Case VI, male, aged forty-nine years. Haematoxylin and eosin stain.  $\times 140$ .

The great majority of nodules in the lung under consideration have apparently originated in such a perivascular lesion. In 17 foci of lymphadenoma in the present section no less than 11 have fairly large blood vessels in or near the centre. In the centre of one focus there is a bronchus, while the remaining five foci are associated with neither bronchi nor blood vessels.

In Case VII there was double hydrothorax, five pints of clear straw-coloured fluid being present in the right pleural cavity and five and a half pints of turbid fluid in the left. The lungs in this case contained many large, ill-defined, tumour-like nodules, some of which were near the hila, others being subpleural.

Half of one such subpleural nodule, as mounted for histological examination, measures 5.0 millimetres from the centre of the central necrotic patch to the periphery. The whole of the transverse diameter of the nodule is 9.5 millimetres.

Microscopically the centre of the nodule is seen to be necrotic. The marginal lymphadenomatous tissue is rich in multinucleated lymphadenoma cells, but the preponderating cell is fibroblastic in type, with a pale vesicular

nucleus which may be ovoid or almost spherical. Fibrillation of cytoplasm is taking place. There is polymorphonuclear leucocytic infiltration, which is most marked in the necrotic part, but present to some extent in the marginal tissue.

The pleura is in contact with part of the nodule. The subpleural fibrous tissue is increased in amount and is only slightly infiltrated by lymphadenomatous tissue, and apparently was not the starting point of the nodule. No large blood vessel or bronchus can be found in the centre of the lesion.

In Case V there is only slight excess of peribronchial fibrous tissue. This is infiltrated by a few hyperchromatic endothelial cells, a few of which are multinucleated. There is no perivascular infiltration.

Simonds, in reviewing the literature, finds that the lung may become involved in one of three ways:

(i) There may be massive invasion of the hilum of the lung from enlarged mediastinal glands. (ii) Extension from the same source along the interlobular lymphatics may produce radiating bands of lymphadenomatous tissue in the lung. (iii) Small isolated nodules may be scattered throughout the lung.

In all three cases referred to above the lymphadenomatous nodules were apparently isolated and scattered, so that they would fall into the third class of Simonds's classification. In a series of five cases reported by MacCallum<sup>(17)</sup> in 1907 the lungs of two contained scattered nodules of lymphadenomatous tissue which was found mainly in the alveolar walls, the alveoli being separated and collapsed.

#### Suprarenal Glands.

In Case VI the right suprarenal gland contained a nodule of lymphadenomatous tissue in the medulla. In one section what are apparently the earliest changes are seen in the walls of medullary blood vessels. In the wall of one vessel there is a lymphocytic infiltration in which are occasional reticulum cells with wrinkled nuclear membranes (Figure IX). A few with hyperchromatic nuclei

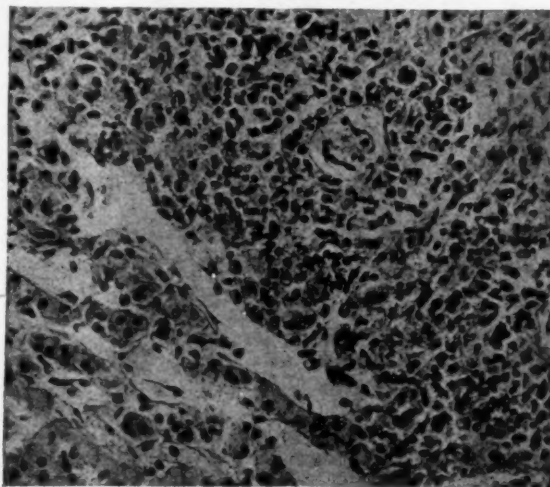


FIGURE IX.

Lymphadenoma tissue in a suprarenal gland. The lymphadenoma tissue is closely associated with a sinusoidal blood vessel. Lymphadenoma cells, hyperplastic reticulum cells and lymphocytes are seen on the right side of the vessel. Suprarenal cortical cells may be seen in the left and lower part of the figure. From Case VI, male, aged forty-nine years. Haematoxylin and eosin stain.  $\times 200$ .

are also present. In the adventitia of an adjacent arteriole a similar change is seen (Figure X), but here there is one cell with two hyperchromatic nuclei; reticulum cells are more numerous and lymphocytes are relatively diminished in number. In other foci there are more numerous and typical lymphadenoma cells.

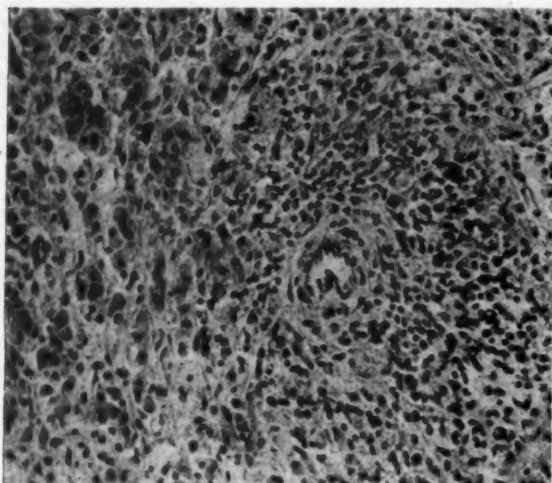


FIGURE X.

Lymphadenoma tissue in a suprarenal gland. The lymphadenoma tissue is seen round a small arteriole. On the left are some suprarenal cortical cells. From Case VI, male, aged forty-nine years. Hematoxylin and eosin stain.  $\times 180$ .

In another section the volume of the medulla is enormously increased. It consists very largely of large cells with abundant pale, foamy cytoplasm and small vesicular nuclei. The whole is supported by a fine capillary stroma with only small areas in which fibrous tissue is recognizable. Here and there, especially near one large sinus, there is definite lymphadenomatous tissue with multinucleated lymphadenoma cells, necrosis, polymorphonuclear leucocytic infiltration and a moderate amount of fibrosis. In one part it is certain that this change has occurred in the cortex and that some of the foamy cells are suprarenal cortical cells. In sections showing earlier changes, however, these are almost entirely confined to the medulla of the gland.

Involvement of the suprarenal glands in Hodgkin's disease is not common. Barron had one case in a series of 24, but Simonds was able to cite several examples from the literature.

#### Note on the History of Hodgkin's Disease.

The original description of the disease by Hodgkin<sup>(18)</sup> in 1832 was referred to by Bright<sup>(19)</sup> in 1838 in a paper on "Abdominal Tumours". Apart from this, Hodgkin's paper appears to have been entirely neglected for many years. In 1856 Wilks<sup>(20)</sup> independently described the affection and more or less completely separated it from lardaceous disease, with which it was frequently confounded. He described the characteristic distribution of the diseased glands, their large size, the absence of fusion of separate glands, their elastic and tough consistence and translucent appearance. He found that the glands were quite unaffected by iodine, thus differing from those of true lardaceous disease. The occasional occurrence of a central area of necrosis was also noted. Microscopic structure could

not at that time be clearly demonstrated, but Wilks described the tissue as being "fibro-nucleated". He added that "sometimes, besides the nucleated fibre element, some transparent, albuminous, amorphous material has been present." This may have referred to the hyaline change so often seen in the fibrous element of the glands. Wilks, from reading Bright's paper, was aware of the existence of Hodgkin's description when he wrote his own, but had been unable to find it until his own paper was completed. When he did find it, he added a footnote disclaiming all credit for his own independent and original description of the disease. In a further paper in 1865 Wilks<sup>(21)</sup> first suggested the name of "Hodgkin's disease".

The same neglect seems to have followed early descriptions of the histology of the disease. Butlin,<sup>(22)</sup> in a discussion at a meeting of the Pathological Society of London in 1902, described the almost general and complete ignorance of the histology of Hodgkin's disease. He himself and others had had great difficulty in diagnosing a case from a section until he recalled a neglected description by Greenfield<sup>(23)</sup> in 1878. He compared his slide with Greenfield's illustrations and found them almost identical. Greenfield had described the thickening of the fibrous stroma, the loss of structure of the gland and the multinucleated cells with four to eight or twelve nuclei. He also noted the later stage of complete fibrosis. To advance the recognition of the pathology of the disease Butlin enlisted the cooperation of Andrewes,<sup>(8)</sup> who at the same meeting of the society read his well known description of its histology. Almost simultaneously Dorothy Reed<sup>(9)</sup> published her classical paper.

#### The Biological Test for Hodgkin's Disease.

Space will not permit any reference to the large volume of work on the aetiology of Hodgkin's disease, but the recent work of M. H. Gordon<sup>(24)</sup> is of unusual interest on account of its bearing on the diagnosis of the disease. Gordon found that if a 10% emulsion of a lymph gland from a case of Hodgkin's disease be injected into the brain of a rabbit, the animal will show symptoms of a meningo-encephalitis after a period of from two to six days. The animal, which exhibits paralysis of the hind limbs, ataxia, muscular rigidity and wasting, may die after three to ten days or may recover.

The gland, which is removed and kept under aseptic conditions, is weighed, minced and ground up, and broth is added to make a 10% suspension. This may be kept for a week in a refrigerator to aid maceration or may be injected immediately. Under anaesthesia a small incision is made through the scalp of the rabbit and with a fine drill a small aperture through the skull is made at a point 2.0 millimetres to one side of the sagittal suture and 1.5 millimetres anterior to the lambdoidal suture. Into the brain 0.4 cubic centimetre is slowly injected and 0.6 cubic centimetre into an ear vein. Out of 20 cases of lymphadenoma, Gordon found that 19 gave positive results, while negative results followed injection of glands from cases of a variety of

diseases, including leucæmia, lymphosarcoma, tuberculosis and carcinoma. Gordon is of the opinion that the active principle in the lymphadenomatous glands is a virus with considerable powers of resistance. The principle is still active after desiccation in a refrigerator for six months and after heating at 65° C. for thirty minutes. It resists treatment with 0.5% carbolic acid for twenty hours at 37° C. and for a fortnight at 0° C. to 6° C. In its powers of resistance Gordon classes the principle with the viruses of vaccinia and psittacosis.

More recently Friedmann<sup>(25)</sup> found that suspensions of normal human bone marrow and spleen, when injected in a similar manner into the brain of a rabbit, produced symptoms similar to those described by Gordon. Friedmann considers that the agent is not a virus, but resembles a proteolytic ferment found by Jochmann in the bone marrow, spleen and leucocytes of man and monkeys. He produced the characteristic encephalitic symptoms in a rabbit by injecting it with human leucocytes.

It cannot be said at present that Friedmann's observations render useless any conclusions drawn from the biological test of lymph glandular tissue. It is of interest to note that suspensions of glands from Gordon's five cases of leucæmia gave negative results.

#### Case Histories.

**CASE I.**—H.B., a male, aged fifty-five years, was admitted to the Royal Prince Alfred Hospital on July 15, 1925. He died on August 20, 1925 (Autopsy Number 4075).

The patient had had dengue eleven, ten and eight years before admission. Each attack was accompanied by swelling of the right inguinal and axillary lymph glands, which later subsided. After an attack six months before admission to hospital the glandular swellings persisted, but diminished in size after taking a mixture containing arsenic. Six weeks before coming to hospital he had an attack of indigestion, followed three weeks later by vomiting and headaches. He became jaundiced six days before admission. The patient had lost 18.9 kilograms (three stone) in weight in six months.

On examination the patient was seen to be moderately jaundiced. There were enlarged, firm, discrete lymph glands in the right posterior triangle of the neck and in the right axilla and both groins. The glands were not fixed to the skin or deeper structures. The liver was palpable five centimetres (two inches) below the costal margin, but the spleen was not palpable.

The patient's temperature was below 37.8° C. (100° F.) during the first six days. During the following seventeen days it was between 37.8° and 40° C. (100° and 104° F.) and in the last fourteen days it varied between 36.1° and 38.1° C. (97° and 100.6° F.).

Blood examination on July 18, 1925, revealed the following information:

Red blood corpuscles, per cubic millimetre	4,310,000
Hæmoglobin value	60%
Colour index	0.69
Leucocytes, per cubic millimetre	5,600
Neutrophile polymorphonuclear cells	64%
Eosinophile polymorphonuclear cells	1%
Lymphocytes	34%
Monocytes	1%

**Autopsy.**—Enlarged lymph glands were found in the right groin. At the root of the right lung was a glandular mass 6.25 centimetres (two and a half inches) in diameter, and a smaller mass was found at the root of the left lung. The glands along the abdominal aorta were enlarged and many of them measured 2.5 by 3.75 centimetres (one by one and a half inches). There was considerable ascites. The spleen was definitely enlarged, it cut with resistance

and showed typical lymphadenoma nodules on section. The liver was slightly enlarged and cut with resistance. On section a fairly advanced grade of cirrhosis was found, but no lymphadenoma was detected. The kidneys, gastrointestinal tract and lungs contained no lesions.

**Microscopic Findings.**—The lymph gland sectioned is very rich in pale hyperplastic reticulum cells with which are a moderate number of lymphadenoma cells. There is early fibrosis, but no necrosis. The spleen contains lymphadenomatous areas which are surrounded by zones of lymphocytes. There is a small amount of necrosis associated with polymorphonuclear leucocytic infiltration. No fibrosis is present. In the liver lymphadenomatous tissue is limited to the enlarged portal canals. There is no necrosis. Some cirrhosis is present apart from the lymphadenoma. There is no lymphadenoma in the kidney.

**CASE II.**—V.H., a male, aged nine years, was first admitted to the Royal Prince Alfred Hospital on May 18, 1925. He died on January 16, 1926 (Autopsy Number 4162).

The patient was well until six months before admission to hospital, when, after a series of severe epistaxes, he became very pale and languid. He had bilious attacks with vomiting and a slight icteric tinge.

Clinical examination revealed slightly doubtful glandular enlargement on the right side of the neck. The liver was enlarged to 2.5 centimetres (one inch) below the costal margin. The spleen was not palpable, but was enlarged to percussion. The temperature was 39.1° C. (102.4° F.) on the day after admission, but fell gradually, becoming normal on the fifth day. The patient was discharged and readmitted three times.

During the second stay in hospital enlarged, tender glands were present in the right side of the neck and the spleen became palpable. A gland in the neck was fomented and discharged pus. The temperature was normal at first, but rose to 38.9° C. (102° F.) on the tenth day and afterwards fell slowly, the evening temperature being normal on the seventeenth day.

During the third stay in hospital a right inguinal gland, which had become enlarged, was removed for examination. During this period the temperature was at first normal, but the evening temperature rose to 37.9° C. (100.2° F.) on the eleventh day and 39.1° C. (102.4° F.) on the thirteenth day. The evening temperature continued at about this level till the sixteenth day, after which it remained in the neighbourhood of 37.8° C. (100° F.), falling to normal in the morning.

During the last period in hospital there was generalized œdema involving the face, ankles and genitals. Ascites was present and *paracentesis abdominis* was performed three times. The urine was very scanty and contained "almost solid albumin on boiling". He had diarrhoea three days before death. The evening temperature varied from 37.2° to 38.2° C. (99° to 100.8° F.), but was subnormal during the last three days.

**Autopsy.**—A few ounces of straw-coloured fluid were found in the general peritoneal cavity. Enlarged lymph glands were present in the right inguinal region and in the abdomen, especially round the origin of the superior mesenteric artery, along the abdominal aorta and in the portal fissure. A few of the glands showed small hæmorrhages. The spleen was enlarged, firm and dark, and typical "suet nodules" were seen on section. Some of these measured two centimetres in diameter and had the colour of splenic pulp, but others were pale and contained small hæmorrhages. The liver showed mottling on the surface. On section there were some small pale areas which were most noticeable near the capsule. Other parts of the liver showed chronic passive venous congestion. The kidneys weighed 115 grammes each. The capsules were not adherent and the surfaces were smooth. The cortices had a yellowish white colour and a somewhat granular texture. The pyramids were engorged. The gastrointestinal tract and the suprarenal glands showed no evidence of Hodgkin's disease.

**Microscopic Findings.**—The lymph gland sectioned is almost entirely hyaline, but contains some more cellular areas in which a few lymphadenoma cells are present.

There is no necrosis and no polymorphonuclear leucocytic infiltration. The capsule is thick, but not infiltrated. In the spleen there are many fibrous areas containing scattered lymphadenoma cells as well as some areas of necrosis associated with slight polymorphonuclear leucocytic infiltration. In the liver there is lymphadenomatous tissue in the portal canals. It shows no necrosis and only slight fibrosis. There is no hyperplasia of Kupffer cells. No lymphadenomatous tissue is found in the kidneys, but practically all glomeruli are enlarged and show a change which appears to be similar to the "focal necrosis" described by Russell. No definite adhesions of tuft to capsule are seen, but the tuft fills the capsule in most instances. There is toxic spoiling of the epithelium of the convoluted tubules.

CASE III.—L.N., a male, aged five years, was admitted to the Royal Prince Alfred Hospital on July 26, 1930. He died on October 10, 1930 (Autopsy Number 5438).

The patient, who had had enlarged cervical glands since the age of fifteen months, was brought to hospital on account of swelling of the abdomen.

The child was sick-looking and pale, the skin having a lemon tint. On the left side of the neck there was a large mass, 11.25 centimetres (four and a half inches) in diameter, formed of firm, discrete glands, which were freely movable on each other and not tender. Several smaller glands were felt on the right side of the neck, as well as in both axillae. The abdomen was full, but no tenderness was detected. The spleen and liver were palpable. While in hospital the patient had X ray therapy. Some days before death he developed dyspnoea with "wheezing".

Blood examination on July 29, 1930, revealed the following information:

Red blood cells, per cubic millimetre ..	2,530,000
Hæmoglobin value .. .. .	30.0%
Colour index .. .. .	0.59
Leucocytes, per cubic millimetre .. ..	3,160
Neutrophile polymorphonuclear cells ..	79.5%
Eosinophile polymorphonuclear cells ..	1.0%
Lymphocytes .. .. .	16.5%
Monocytes .. .. .	3.0%

Anisocytosis, poikilocytosis and polychromasia were pronounced. Seven megaloblasts occurred among 200 leucocytes.

**Autopsy.**—Enlarged cervical lymph glands, especially on the left side, extended from above the clavicle to the base of the skull. Enlarged glands were also found in the axillae and in the anterior, superior and posterior mediastina. There were numerous large lymph glands in the abdomen, those specially noted being situated along the abdominal aorta, between the kidneys, along the superior border of the pancreas, at the hilum of the spleen and in the hepato-duodenal ligament. Smaller glands were also noted in the inguinal regions. The lymph glands were discrete and on section were pale, slightly greyish and semi-translucent, with some opaque cream-coloured areas. The spleen was very much enlarged and reached to the level of the umbilicus. The cut surface exhibited many lard-like areas one to three millimetres in diameter. The lungs, liver, kidneys and suprarenal glands showed no evidence of Hodgkin's disease.

**Microscopic Findings.**—An inguinal lymph gland shows very early changes. The architecture is distorted, but some dilated lymph sinuses are still present and the gland is highly vascular. There is patchy overgrowth of reticulum cells, but fibrosis is very slight. Occasional lymphadenoma cells are found. Lymphocytes are much diminished in number. In an iliac lymph gland the overgrowth of the reticulum cells is much more pronounced than in the inguinal gland just examined. Lymphadenoma cells are more numerous and more fibrosis is present. There are small patches of necrosis. In a lymph gland from near the base of the skull there is pronounced fibrosis, the fibrous tissue having a reticulated or coarsely foamy appearance. Lymphocytes are very much reduced in number. Typical lymphadenoma cells are present, but are hard to find. A gland from the mediastinum consists mainly of dense hyaline fibrous tissue, but has more

cellular areas where the fibrosis is coarsely foamy, here resembling the gland from the base of the skull. A bronchial gland is similar to that from the mediastinum. The spleen contains small and large foci of lymphadenoma. Some show advanced fibrosis, but many are fibro-cellular. There are small areas of necrosis. No lymphadenomatous tissue is found in sections from the liver and kidneys.

CASE IV.—J.C., a male, aged forty-four years, was admitted to the Royal Prince Alfred Hospital on April 23, 1931. He died on September 23, 1931 (Autopsy Number 5694).

The patient had had a cough and pain in the left side of the chest for seven months. Seven weeks before his admission to hospital enlarged lymph glands were discovered in the left side of the neck. One was excised for examination at another hospital and a course of deep X ray therapy was commenced. About eight days before admission attacks of coughing were followed by vomiting and pain in the chest. Dyspnoea, orthopnoea and headache were experienced. The patient lost 15.7 kilograms (two and a half stone) in weight in seven months.

On examination small lymph glands were palpable in the groins, one gland was felt in the right axilla, and in the left posterior triangle of the neck there was a fairly large rubbery-hard gland which was not fixed to the skin and was not tender. The abdomen was slightly protuberant; the edge of the liver could be felt 6.75 centimetres (two and a half inches) below the costal margin, and the spleen was palpable.

Blood examination on April 23, 1931, revealed the following information:

Red blood cells, per cubic millimetre ..	3,900,000
Hæmoglobin value .. .. .	56.0%
Colour index .. .. .	0.71
Leucocytes, per cubic millimetre .. ..	3,540
Neutrophile polymorphonuclear cells ..	64.0%
Lymphocytes .. .. .	34.5%
Monocytes .. .. .	1.5%

After being in hospital for nearly three months the patient was allowed to go home, but a week later he was readmitted.

Reexamination of the patient at this time (July 15, 1931) revealed many small supraclavicular glands. The edge of the liver was felt 2.5 centimetres (one inch) below the costal margin, and the spleen was much enlarged.

A further blood examination on July 20, 1931, revealed the following information:

Red blood cells, per cubic millimetre ..	2,850,000
Hæmoglobin value .. .. .	61%
Colour index .. .. .	1.07
Leucocytes, per cubic millimetre .. ..	2,880
Neutrophile polymorphonuclear cells ..	20%
Eosinophile polymorphonuclear cells ..	3%
Lymphocytes .. .. .	66%
Monocytes .. .. .	11%

**Autopsy.**—The skin was somewhat pigmented. Small "shotty" lymph glands were noted in the groins. Slightly enlarged "rubbery" glands were found in the neck, along the abdominal aorta and round the head of the pancreas, but they were nowhere very large. Bronchial glands were anthracotic and calcified, but showed no evidence of lymphadenoma. The spleen was enlarged (450 grammes) and there were fine fibrous adhesions to surrounding structures. The capsule was a little thickened and on section there were many small "suet" nodules, each about two or three millimetres in diameter. The liver was much enlarged. One small white area was noted under the capsule. Other organs showed no special abnormality.

**Microscopic Examination.**—In an inguinal lymph gland there is gross reticulum cell hyperplasia with some fibrosis, which is most marked in the centre of the gland. The gland is highly cellular and lymphadenoma cells are numerous. A few small areas of necrosis are present. In a cervical lymph gland there is much more fibrosis, but the gland is still very cellular. Lymphadenoma cells are numerous. A gland from near the pancreas is much more fibrous, but there are some more cellular areas in which lymphadenoma cells are present. In a lymph gland from

near the aorta the greater part is hyaline, but a few small cellular areas remain.

One section of the spleen shows very widespread necrosis suggesting the presence of an infarct. In another there are many small areas of lymphadenomatous tissue, some of which are necrotic. Other foci show considerable fibrosis. Lymphadenoma cells are not very numerous. In the liver lymphadenomatous tissue is found in the portal canals. Here there are very numerous lymphadenoma cells and a large amount of necrosis. The Kupffer cells are hyperplastic and pigmented.

CASE V.—R.F., a male, aged twenty-five years, was admitted to the Royal Prince Alfred Hospital on February 6, 1933. He died on May 22, 1933 (Autopsy Number 6048).

About two years before admission to hospital lymph glands in the patient's neck became enlarged and the enlargement was thought to be due to tonsillitis. The swelling in the neck became larger and two months later the glands in other parts of the body became swollen. He was admitted to another hospital, where a gland was excised for examination, and after treatment by X rays the swellings subsided. After this the patient was in good health until two months before admission, when he experienced an attack of shivering and had a high temperature with general debility, which lasted for several weeks. Nearly two months later he had a similar attack. There was a history of syphilis contracted three years previously. The patient was treated by injections of an arsenical preparation and later developed a rash, which was considered to be an arsenical dermatosis.

On physical examination the patient was seen to be a pale young man. Enlarged lymph glands were present in the neck, including the right supraclavicular region, in both axillae and both groins. The abdomen was tender on pressure in the left hypogastrium. The spleen was not palpable, but on percussion was thought to be enlarged. The liver dullness was normal in extent. No glandular enlargement in the thorax was detected on X ray examination.

Blood examination on February 7, 1933, revealed the following information:

Red blood cells, per cubic millimetre ..	4,370,000
Hæmoglobin value .. .. .	76.0%
Colour index .. .. .	0.86
Leucocytes, per cubic millimetre .. ..	5,100
Neutrophile polymorphonuclear cells ..	59.0%
Lymphocytes .. .. .	31.5%
Monocytes .. .. .	9.5%

The Wassermann test gave no reaction.

While in hospital the patient had several periods of pyrexia, during which the temperature was in the neighbourhood of 39.4° and 40° C. (103° and 104° F.). These lasted about one week and recurred at intervals of about five weeks. The superficial glands never became enlarged, but the spleen became palpable and the liver enlarged and tender. His anæmia became progressively worse until on May 16, 1933, examination of the blood showed:

Red blood cells, per cubic millimetre ..	1,730,000
Hæmoglobin value .. .. .	34%
Colour index .. .. .	0.98
Leucocytes, per cubic millimetre .. ..	2,300
Neutrophile polymorphonuclear cells ..	67%
Lymphocytes .. .. .	22%
Monocytes .. .. .	11%

Anisocytosis and poikilocytosis were present.

**Autopsy.**—A few enlarged, discrete, rubbery lymph glands were found in the neck, the largest being in the upper left deep cervical group. A few small lymph glands were noted in the groins. Enlarged lymph glands were found in the abdomen, along the abdominal aorta and iliac arteries as far as Poupart's ligaments. No enlarged glands were found in the thorax.

The spleen was very much enlarged, weighing 540 grammes. The capsule showed patchy thickening and on section there were numerous white flecks which suggested early lymphadenomatous involvement. The liver was slightly enlarged, weighing 1,670 grammes. It showed some fine marbling immediately under the capsule on the posterior aspect.

**Microscopic Examination.**—In the lymph glands there is gross overgrowth of reticulum cells, most of which are hyperchromatic. Lymphadenoma cells are numerous. Fibrosis is present, but is not very pronounced. Necrosis is well marked in two lymph glands, but very slight in a third.

In the spleen definite lymphadenoma cells are scarce. There is overgrowth of reticulum cells which resemble lipid histiocytes. One small area of necrosis is seen in the section, as well as some areas of fibrosis with early hyaline change. A section of the liver contains one area of lymphadenoma, which has apparently originated in a portal canal near the surface of the liver. One other canal shows very early lymphadenoma, while others are infiltrated by lymphocytes. A section of the lung shows peribronchial fibrosis, in which there are lymphocytes and some hyperchromatic reticulum cells. A few lymphadenoma cells are present, but these are not isolated from neighbouring cells. Some foci consist only of lymphocytes embedded in peribronchial fibrous tissue.

CASE VI.—D.W., a male, aged forty-nine years, was admitted to the Royal Prince Alfred Hospital on January 1, 1934. He died on January 8, 1934 (Autopsy Number 6207).

About eighteen months before admission to hospital the patient first noticed a lump in the right groin. He was treated by deep X ray therapy and the lump rapidly subsided. Since then he felt well until three weeks ago, when he suffered from a severe headache and loss of appetite. He was found to have an elevated temperature and was sent to hospital.

On physical examination slight jaundice was noted. The liver was enlarged to 7.5 centimetres (three inches) below the costal margin, but no note concerning the spleen or superficial lymph glands has been preserved. Three days after admission he was more jaundiced, and two days later he was comatose. The jaundice later increased until the skin became the colour of bronze.

Blood examination on January 6, 1934, revealed the following information:

Red blood cells, per cubic millimetre ..	4,270,000
Hæmoglobin value .. .. .	74.0%
Colour index .. .. .	0.86
Leucocytes, per cubic millimetre .. ..	9,000
Neutrophile polymorphonuclear cells ..	59.0%
"Band-form" polymorphonuclear cells ..	22.0%
Lymphocytes .. .. .	13.5%
Monocytes .. .. .	5.5%

One normoblast was seen in the film.

During the first five days the temperature ranged between 38.9° and 40° C. (102° and 104° F.). It then fell to the vicinity of 38.3° C. (101° F.), where it remained during the last three days.

**Autopsy Report.**—There was deep jaundice of the skin and conjunctivæ. The lymph glands in the groins were enlarged, rubbery and discrete, as were those along the abdominal aorta. On section they were seen to be pale and mainly translucent, but they contained some opaque, yellow areas of necrosis. The lungs were large, pale and emphysematous and felt nodular throughout. The right lung weighed 530 grammes and the left 440 grammes. On section there were numerous pale nodules, the size of a split pea, scattered throughout the lung substance. They were not obviously related to blood vessels or to bronchi. The spleen was unusually large and globular. Many prominent, pale, firm areas were seen on the surface and on section there were many pale areas which were well defined and varied in size from one to three millimetres in diameter. The liver was much enlarged and had a blunt anterior margin. Superficially many pale tumour-like nodules projected slightly from the surface; one was slightly umbilicated. On the cut surface numerous tumour-like nodules were seen to surround large veins, some of which were obviously branches of the portal vein and others tributaries of the hepatic vein. The nodules varied in size from one to three centimetres in diameter. In the medulla of the right suprarenal gland there was a tumour-like nodule. The heart, kidneys and gastro-intestinal tract showed no lesions.

**Microscopic Examination.**—In the lymph glands there are, in two of the sections, many areas of necrosis with lymphadenomatous tissue between them. There are definite lymphadenoma cells in many parts, but there are also unusually large numbers of cells which have taken up brown blood pigment. Polymorphonuclear infiltration is found in and around the necrotic areas. Two other lymph glands show wide areas of hyaline tissue with some perivascular lymphoid accumulations and scattered phagocytic cells which are loaded with brown blood pigment. No lymphadenoma cells are seen in these two sections. In the spleen there are large areas of lymphadenomatous tissue which are necrotic and are infiltrated by polymorphonuclear leucocytes. Other parts are almost completely necrotic, but here the nuclei of lymphadenoma cells retain their selective staining with hæmatoxylin. Fibrosis is mainly perivascular and is associated with cells containing brown blood pigment. The more normal areas of the section are infiltrated with polymorphonuclear leucocytes. In the liver there are very large ill-defined masses of lymphadenomatous tissue which contain large areas of necrosis. There is very little fibrosis. In the less affected parts the Kupffer cells are hyperplastic and loaded with brown blood pigment. In another section smaller but well advanced lymphadenomatous changes are found in the portal canals. Numerous large lymphadenomatous areas are found in the lungs. These are mainly situated round blood vessels and contain large areas of necrosis. They are more fully described above. The right suprarenal gland, which contains a large area of lymphadenomatous tissue, is also fully described in the paper.

CASE VII.—O.J., a male, aged twenty-six years, was admitted to the Royal Prince Alfred Hospital on June 26, 1932. He died on January 19, 1934 (Autopsy Number 6225).

About eight months before coming under observation the patient began to have copious night sweats. He lost 6.3 kilograms (one stone) in weight, developed a soreness in the neck and noticed a swelling in that part. Later he began to lose strength and became breathless on exertion. More recently his voice became husky and he developed a cough with copious frothy sputum. There was some frequency of micturition and diarrhoea. In the last two weeks the tips of the fingers became swollen.

On examination the patient was seen to be a fairly well nourished but pale young man. The tips of the fingers were clubbed. The lymph glands on both sides of the neck were swollen and soft. They were not matted together, but were movable on each other. Those on the left side were tender. Two or three small soft lymph glands were found in each axilla. No glands were palpable in the groins. X ray examination revealed enlarged glands in the hila of both lungs, enlargement of the paratracheal glands, and some irregular infiltration towards both bases. Examination of the abdomen revealed no abnormality. The spleen and liver were not enlarged.

Blood examination on June 27, 1932, revealed the following information:

Red blood cells, per cubic millimetre ..	4,260,000
Hæmoglobin value .. .. .	66%
Colour index .. .. .	0.77
Leucocytes, per cubic millimetre .. ..	15,400
Neutrophile polymorphonuclear cells ..	92%
Lymphocytes .. .. .	7%
Monocytes .. .. .	1%

Slight polychromasia was present.

The patient was treated by deep X ray therapy, after which his condition improved and the enlargement of the superficial glands disappeared. He was discharged and readmitted several times.

On April 6, 1933, he had lost 4.5 kilograms (ten pounds) in weight since being discharged eight months previously. Enlarged lymph glands were present in the axillæ and low down on the left side of the neck. The liver and spleen were not enlarged.

During his first period in hospital (June 26, 1932, to August 14, 1932) there was fairly continuous pyrexia up to 38.3° and 38.9° C. (101° and 102° F.) for eight days. The temperature then fell by lysis over a period of

seven days, and the remainder of this stay in hospital was apyrexial. During the second period (April 6, 1933, to June 19, 1933) there was irregular pyrexia in the neighbourhood of 37.8° C. (100° F.).

Five days after being discharged the patient died at his home, but the body was brought to hospital for autopsy.

**Autopsy.**—The abdomen was slightly distended, but the arms and legs were wasted. Enlarged lymph glands were found in the left side of the neck, in the right axilla and at the hila of the lungs. A large retroperitoneal mass of glands was found in the upper part of the abdomen. These were partly semi-translucent on section and partly pale and necrotic. They extended along the body of the pancreas and had pushed the stomach forward; they were adherent to that viscus and lymphadenomatous tissue had invaded the stomach wall. Large soft glands were also present in the *porta hepatis* and hepato-duodenal ligament.

The right pleural cavity contained five pints of clear straw-coloured fluid, and five and a half pints of turbid fluid were present in the left pleural cavity. A slight excess of fluid was found in the peritoneal cavity. The lungs contained many tumour-like nodules of lymphadenoma tissue, which measured up to one centimetre in diameter. Some of the nodules were situated near the hila, while others were subpleural. The lungs were collapsed posteriorly and slightly emphysematous anteriorly.

The spleen was somewhat enlarged (240 grammes), but showed no evidence of Hodgkin's disease. Malpighian bodies were not prominent. The liver was enlarged and weighed 1,830 grammes. It contained many tumour-like masses, mainly on the anterior surface. A large tumour-like mass had almost entirely replaced the caudate lobe. Large soft lymph glands were found, as noted above, at the *porta hepatis* and in the hepato-duodenal ligament. Several round tumour-like nodules were found in the left kidney, which weighed 170 grammes. The right kidney weighed 130 grammes and contained no deposits of lymphadenoma tissue.

The stomach, as mentioned above, was pushed forward by the large glandular mass associated with the pancreas. It was adherent to the mass and lymphadenomatous tissue had invaded the stomach wall in the region of the lesser curvature near the cardia. The invaded portion was ulcerated, the ulcer being six centimetres in diameter, and its base being formed of lymphadenoma tissue. The edges were infiltrated and in places everted. No lymphadenomatous tissue was found in the intestines.

**Microscopic Findings.**—Sections of the lymph glands show, in one instance, a large central area of necrosis. The tissue surrounding this zone is fibrillar, while the marginal parts are more cellular but less fibrous. Lymphadenoma cells are numerous in the more cellular parts. Other glands show similar changes.

The spleen shows no microscopic evidence of lymphadenoma. It is very much engorged and there is an excess of polymorphonuclear leucocytes in the pulp between the sinusoids. Very small arterioles with hyperplastic endothelium are surrounded by small foci of plasma cells with which are occasional polymorphonuclear cells.

The liver contains a large and fairly well defined tumour-like mass of lymphadenomatous tissue. This is very cellular and includes hyperplastic reticulum cells, some of the nuclei of which are pale and vesicular and some hyperchromatic. Well defined lymphadenoma cells with multiple nuclei and deeply stained nucleoli are also found. Some of the cells are enlarged and fibroblastic, yet fibrosis is at a minimum. The surrounding liver tissue is compressed and shows considerable engorgement. The Kupffer cells are pigmented and slightly hyperplastic. The portal canals contain no lymphadenomatous tissue, but are infiltrated by polymorphonuclear leucocytes.

The section from the stomach was taken from the edge of the ulcer and includes the mucosa on one side and the muscularis on the other. The submucosa is much thickened by the presence of a mass of lymphadenoma tissue which contains the usual variety of cells, including lymphadenoma cells. The side of this tissue which formed the edge of the ulcer is necrotic and infiltrated by polymorphonuclear cells. The lymphadenoma tissue comes into contact with the mucosa at one point and on the deeper side it infiltrates

TABLE III.  
Summary of Blood Counts in Eight Cases of Hodgkin's Disease.

Case Number.	Date.	Red Blood Corpuscles per Cubic Millimetre in Millions.	Hæmoglobin per centum.	Colour Index.	Total Leucocytes per Cubic Millimetre.	Polymorphonuclear Leucocytes.			Lymphocytes per centum.	Monocytes per centum.
						Neutrophile per centum.	Eosinophile per centum.	Basophile per centum.		
I	18/7/25	4.31	60	0.69	5,600	64.0	1.0	—	34.0	1.0
	3/8/25	5.66	70	0.61	10,200	60.0	1.0	—	37.0	2.0
	20/8/25	3.3	50	0.75	10,400	80.5	—	0.5	17.5	1.5
II	21/5/25	5.0	60	0.6	7,200	60.0	—	1.0	38.0	1.0
	22/6/25	4.9	70	0.71	8,500	69.0	1.0	—	28.0	2.0
	12/10/25	3.88	38	0.48	4,800	51.0	1.0	0.5	39.0	8.5
	6/1/26	4.53	60	0.66	9,600	70.0	—	—	28.5	1.5
III	29/7/30	2.53	30	0.59	3,160	79.5	1.0	—	16.5	3.0
	5/9/30	2.01	22	0.54	5,360	82.0	2.0	—	13.0	3.0
IV	23/4/31	3.9	56	0.71	3,540	64.0	—	—	34.5	1.5
	18/5/31	2.85	55	0.96	6,600	9.0	—	—	82.0	9.0
	23/6/31	2.06	45	1.09	3,480	62.0	1.5	1.5	28.5	6.5
	20/7/31	2.85	61	1.07	2,880	20.0	3.0	—	66.0	11.0
	4/8/31	—	—	—	4,340	61.5	—	—	28.0	10.5
	25/8/31	3.1	52	0.83	2,160	60.0	—	—	31.0	9.0
	2/9/31	2.31	36	0.77	1,180	60.0	—	—	28.0	12.0
	7/9/31	2.48	35	0.7	1,180	60.0	—	—	32.0	8.0
	22/9/31	1.15	18	0.78	2,280	78.0	—	—	16.0	6.0
V	7/2/33	4.37	76	0.86	5,100	59.0	—	—	31.5	9.5
	23/2/33	4.23	76	0.89	4,600	61.0	—	1.0	31.0	7.0
	28/2/33	4.16	74	0.88	3,700	68.0	—	—	30.0	2.0
	6/3/33	4.12	75	0.91	2,200	32.0	—	—	64.0	4.0
	5/4/33	3.97	74	0.93	2,200	56.0	—	—	35.0	9.0
	26/4/33	2.31	43	0.93	2,400	54.0	—	—	36.0	10.0
	2/5/33	—	—	—	2,600	60.0	—	—	32.0	8.0
	11/5/33	2.11	40	0.94	1,300	68.0	—	—	25.5	6.5
	16/5/33	1.73	34	0.98	2,300	67.0	—	—	22.0	11.0
VI	6/1/34	4.27	74	0.86	9,020	81.0	—	—	13.5	5.5
VII	27/6/32	4.26	66	0.77	15,400	92.0	—	—	7.0	1.0
	11/7/32	3.74	60	0.80	23,700	95.0	—	—	4.0	1.0
	26/7/32	4.80	80	0.83	6,020	82.0	0.5	—	15.5	2.8
	4/8/32	4.93	82	0.83	4,100	80.0	5.0	1.0	14.0	—
	11/8/32	4.73	80	0.84	5,020	78.0	5.0	2.0	9.5	5.5
	7/4/33	4.62	92	0.99	6,700	83.0	2.5	1.0	9.0	4.5
	22/5/33	4.52	88	0.97	3,300	83.5	2.0	1.0	10.5	3.0
	9/6/33	4.56	90	0.98	3,800	82.0	2.5	1.5	9.0	5.0
	8/12/33	3.74	66	0.88	7,100	85.0	1.5	—	8.5	5.0
VIII	12/11/24	—	—	—	5,700	—	—	—	—	—
	19/11/24	5.5	72	0.65	6,000	69.0	—	—	5.0	26.0
	29/11/24	4.0	60	0.75	4,700	—	—	—	—	—
	3/12/24	—	—	—	8,000	70.0	—	—	4.0	26.0
	5/12/24	—	—	—	6,000	73.0	—	—	2.0	25.0

and partly destroys the muscularis. A small isolated area of lymphadenomatous tissue is present in the submucosa at a distance from the main mass.

A nodule from the lung shows the presence of a central area of necrosis which is surrounded by a highly cellular area. The predominating cell is fibroblastic in type, but many lymphadenoma cells are also present.

CASE VIII.—J.W., a male, aged thirty-two years, was admitted to the Royal Prince Alfred Hospital on November 12, 1924. He died on December 14, 1924 (autopsy report not numbered).

Four and a half years before admission to hospital the patient had pneumonia, which followed an attack of influenza. After being in bed for three weeks he was about to get up when he had a further period of pyrexia, which continued on and off for ten weeks. This was accompanied by weakness, malaise, diarrhoea and jaundice. The latter symptom occurred at intervals during the last few weeks.

On examination the patient was seen to be thin and sallow. His facies suggested typhoid fever. The abdomen was slightly distended and the liver somewhat enlarged.

No enlargement of the spleen was detected at this time, but some rigidity of the upper part of the left rectus abdominis muscle was noted. Enlarged lymph glands were first observed in the posterior cervical region sixteen days after his admission to hospital, and four days later the spleen was palpable.

Blood examination on November 19, 1924, revealed the following information:

Red blood cells, per cubic millimetre ..	5,500,000
Hæmoglobin value .. .. .	72%
Colour index .. .. .	0.65
Leucocytes, per cubic millimetre .. ..	6,000
Neutrophile polymorphonuclear cells ..	69%
Lymphocytes .. .. .	5%
Monocytes .. .. .	26%

The jaundice diminished in intensity for a time, but later became more marked. Bile was present in the urine and the stools were clay coloured.

The patient's temperature was 39.1° C. (102.4° F.) on admission, but it became almost normal within three days. A period without pyrexia then followed and lasted nine days. After that the temperature gradually rose and

remained between 38.3° and 39.4° C. (101° and 103° F.), except for occasional irregularity; until the patient died four and a half weeks after admission to hospital.

**Autopsy.**—Slightly enlarged lymph glands were found on the right side of the neck, and the mediastinal glands were somewhat enlarged. Many large glands were found along the abdominal aorta, and those round the pancreas were the size of a pigeon's egg.

The spleen was slightly enlarged and was firm in consistency. On section numerous small yellowish nodules were seen scattered through the parenchyma. In the liver were many irregular areas of yellowish tissue surrounding the bile channels and blood vessels.

**Microscopic Examination.**—Lymph gland is replaced by lymphadenomatous tissue, which in some parts is highly cellular and contains some lymphadenoma cells. In other places fibrosis is more abundant, but lymphadenoma cells are still prominent. There are areas of necrosis which are thickly infiltrated by polymorphonuclear leucocytes. At the edge of another gland the fat is being invaded by lymphadenomatous tissue. In this gland necrosis is much more massive. In the spleen are lymphadenomatous areas with fibrosis and necrosis. In non-necrotic foci lymphadenoma cells are prominent. In the liver are many lymphadenomatous areas. The smaller foci have commenced in the portal canals. Necrosis is present in some larger foci.

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## Reports of Cases.

### TETANY COMPLICATED BY CARDIAC FAILURE.

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AND

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THE patient, a married woman of thirty-eight years, was admitted to hospital complaining of increasing dyspnoea, swelling of the feet, palpitation and fatigue, which had been coming on for the last year and which were becoming more severe. She complained of what she described as "attacks of paralysis in the hands and feet", which she had had at intervals for ten years and which were becoming more frequent and more severe, now occurring about two or three times a day.

She had four living children and at each confinement had bled excessively. She had one miscarriage at the fourth month, attended also by much bleeding. She had a hysterectomy performed at the age of thirty-one for menorrhagia. She had a severe attack of scarlet fever at the age of twenty-nine, after which she was obliged to stay in bed for six weeks.

Physical examination revealed a thin, pale woman with slight dyspnoea while at rest in bed. Her pulse rate was 126 in the minute, its volume and tension were poor, the pulse was regular. The cardiac dullness extended 1.25 centimetres (half an inch) to the right of the sternum, while the apex beat was in the sixth interspace, 11.25 centimetres (four and a half inches) from the mid-sternal line. There was an apical systolic murmur conducted out to the axilla. The systolic blood pressure was 126 and the diastolic pressure 90 millimetres of mercury. The lungs were clear. Examination of the abdomen revealed no abnormality. There was moderate oedema of the ankles. The urine contained a moderate amount of albumin, but was otherwise normal. A blood count (made a fortnight after admission) showed a red count of 3,890,000 per cubic millimetre, the picture being that of a secondary anaemia.

Shortly after admission to hospital the patient was seen to become rigid, the hands assuming the "main d'accoucheur" position, with flexion of wrists and elbows and adduction of arms; the legs were extended, with plantar flexion of ankles and toes. The patient was unable to articulate. The pulse rose in the attack to 160 and was almost imperceptible. The attack lasted about six and a half minutes and then the spasm rapidly lessened. The patient afterwards described a choking sensation in the throat and a feeling of constriction in the chest, together with severe cramp-like pains in the upper part of the abdomen. She said that she always felt as if she might die of suffocation in such an attack. It was possible to elicit Trousseau's sign with the sphygmomanometer, but Chvostek's sign was not obtained.

The patient was given tincture of digitalis in full doses and later, when fully under the influence of the drug, a maintenance dose of 0.48 mil (eight minims) three times

daily was continued. She was given calcium chloride, 0.12 gramme (two grains), in a 5% solution intramuscularly every four hours, and parathyroid extract, 0.006 gramme (one-tenth of a grain), was given by mouth at the same time. Under this treatment she had four spasms on the first day, two on the second day, and none thereafter for over a fortnight.

After the first week the calcium chloride was discontinued and calcium lactate was given by mouth, the patient taking 3.6 grammes (sixty grains) a day. The parathyroid extract was continued, 0.006 gramme (one-tenth of a grain) being given three times a day. She had four further spasms during the five weeks she was in hospital, but all were slight and transient.

The oedema and albuminuria had disappeared and no increase in the cardiac dullness above normal was demonstrable after she had been five weeks in bed. The patient was then permitted to go home, but was kept on the maintenance dose of digitalis, and calcium and parathyroid in the quantities quoted above.

Her condition continued to improve and after two months she was able to dress herself and move from room to room, and it was proposed to send her to Adelaide for biochemical investigation. She then contracted a prevalent influenza in a severe form and a week after the onset she began to get slight tetanic spasms—two to three daily. She improved for two days with intramuscular injections of calcium, but on the third day got a severe spasm lasting about fifteen minutes; she became afterwards very short of breath, and the pulse rate rose to 130, and the next day she had cardiac dullness 2.5 centimetres (one inch) to the right of the sternum, and the apex beat was in the sixth interspace, 10.0 centimetres (four inches) from the mid-sternal line. Oedema of the feet and albuminuria were also present.

The spasms became more frequent and the patient had seven that day. It was found impossible to control them by intramuscular or intravenous injections of calcium, probably owing to the poor state of the circulation. In spite of the cardiac failure present, morphine was given in an attempt to control the spasms, which were exhausting the patient. However, the patient sank and died on the next day, being comatose for the last four hours.

#### Conclusions.

1. The aetiology of the tetany is obscure, though the spasms date from the attack of scarlet fever.
2. The evidence seems to point to the cardiac failure being secondary to the tetany, perhaps due to an actual calcium deficiency in the cardiac muscle itself.
3. The previous history of menorrhagia and hæmorrhage at confinements was probably connected with the fault in the calcium metabolism.
4. The initial splendid response to calcium and parathyroid extract should be noted.
5. The effect of the influenza toxin was disastrous.
6. There was a final complete lack of response to calcium and parathyroid extract.

#### STRYCHNINE POISONING WITH RECOVERY.

By THOMAS ROSE, M.B., B.S. (Sydney),  
Senior Resident Medical Officer, Saint Vincent's  
Hospital, Sydney.

MISS X.Y., aged twenty-five years, attempted to commit suicide by swallowing 28.5 cubic centimetres (one ounce) of *liquor strychnine hydrochloride*, that is, approximately 0.24 gramme (four grains) of strychnine hydrochloride. (This amount was verified by the chemist from whom she stole it.)

Half an hour later convulsions commenced and continued for half an hour before she was attended by a private practitioner, who administered 0.015 gramme (one-quarter of a grain) of morphine hypodermically and 7.0 cubic centimetres (two drachms) of *liquor morphine hydrochloride* by mouth and then anesthetized the patient with ether. She was sent to this hospital still anesthetized,

and gastric lavage was performed with four pints of weak potassium permanganate solution two hours after she took the drug. She was given 7.2 grammes (120 grains) of potassium bromide and convulsions recommenced. Under chloroform anaesthesia 0.42 gramme (seven grains) of "Sodium luminal" in 5% solution was injected intravenously; this kept her deeply unconscious for three hours, after which she had another convulsion. She was again anesthetized with chloroform and one gramme of "Sodium evipan" in 10.5 cubic centimetres of distilled water was injected intravenously. This rendered her unconscious for a further three hours, after which she had no more convulsions, except for a slight spasm of both hands, which was easily controlled by 7.2 grammes (120 grains) of potassium bromide given by mouth.

The patient left hospital five days later apparently none the worse for her experience.

#### Comment.

The lethal dose of strychnine is given by Dixon<sup>(1)</sup> as 0.03 to 0.09 gramme (half to one and a half grains) of strychnine hydrochloride, so that this case is remarkable in recovery from such a large amount. Murrell<sup>(2)</sup> reports recovery from 1.2 grammes (twenty grains) of strychnine, but an emetic was administered at once, whereas this patient's treatment did not commence until one hour after she swallowed the drug.

A further point of interest is the successful use of "Sodium luminal" and "Sodium evipan" intravenously.

#### Acknowledgement.

I am indebted to Dr. R. J. Taylor, honorary assistant physician of this hospital, for his permission to report this case.

#### References.

- (1) W. F. Dixon: "A Manual of Pharmacology", Seventh Edition, 1929, page 124.
- (2) Murrell: "What to do in Cases of Poisoning", Thirteenth Edition, 1925, page 208.

## Reviews.

#### A TEXT BOOK OF SURGERY.

THE fact that the well known text book by Romanis and Mitchiner, which comes from the surgical practice of Saint Thomas's Hospital, London, has reached its fifth edition within eight years, is in itself proof that it has filled a need and is also eloquent testimony to its worth.<sup>1</sup> It is printed in two volumes, the classical division of surgery into general and regional being adopted. As regards set up, printing and illustrations, it leaves little to be desired, and as several chapters have been recast and revised, the present edition is an improvement on the previous editions. The work illustrates the point of view of two practical surgeons and bears the stamp of sound British surgery. It is true that there is at times a little imbalance, owing to some important subjects not being allotted sufficient space, while some rare conditions and obsolete methods are given undue importance. This, of course, may be a reflection of the personal experience of the writers or of their school. It would be no loss to the book to eliminate the illustrations and discussion on arterial suture, while there are some other illustrations, such as Figure 26, which could be omitted without detracting from the value of the work. It is somewhat surprising to find that the authors still advocate the use of potassium permanganate crystals in snake bite, in spite of the fact that they are useless. One would like to see a clearer explanation of the nature of surgical shock, based on modern conceptions, and more emphasis laid on the vicious cycles which produce and maintain this condition. It would also be advantageous,

<sup>1</sup> "The Science and Practice of Surgery" by W. H. C. Romanis, M.A., M.B., M.Ch., F.R.C.S., F.R.S., and P. H. Mitchiner, M.D., M.S., F.R.C.S.; Volume I: General Surgery; Volume II: Regional Surgery; Fifth Edition, 1934. London: J. and A. Churchill. Royal 8vo., pp. 799 and 972, with 758 illustrations. Price: 14s. for each volume.

we think, to include a short paragraph on the danger signs in blood transfusion. This would round off what is a very good section. In the treatment of hæmophilia the intramuscular injection of thirty to forty cubic centimetres of normal whole blood is not mentioned. The chapter on post-operative treatment is sound and valuable, while that on bones and joints is excellent. The chapters dealing with fractures have been rewritten and are in the main a very good statement of modern technique. A useful section is that on amputations in which not only the sites of selection and methods are given, but also a short description of artificial limbs. In describing the ætiological factors in hydatid disease, the writers adhere to the erroneous belief that infected drinking water and water cress are important, in spite of the fact that all authorities believe that, as the ova of the *Tania echinococcus* sink in water, this is a relatively unimportant source of human infestation. Direct contact with and handling of infected dogs is undoubtedly the most important factor. The work could be improved by a fuller discussion of Hodgkin's disease and surely the surgery of the sympathetic system deserves more than two pages. The inclusion of chapters on diseases of special organs, which are admirable, being really condensed text books in themselves, adds greatly to the value of the book for students. In the main, the regional sections are the best in the book, those on the abdominal organs being particularly good. That dealing with appendicitis is excellent and no doubt reflects the practice which has been proved at Saint Thomas's. It should be possible in this volume, however, to allow more room for subjects which are of increasing clinical interest, such as the surgery of the lungs, recent advances in surgical pathology of various disease *et cetera*, by omitting some of the text concerning rare conditions which few surgeons see outside big hospitals. We would also suggest that the classical works of other surgeons in various surgical fields should have received fuller acknowledgement. The absence of the names of many great surgeons from the work is somewhat unfair and tends to give students and others the wrong impression. In spite of the above criticisms, however, the work is one of the best of its kind in English; in addition it has been kept relatively up to date, is relatively cheap compared to many others of the same type and can be confidently recommended to all students and graduates.

#### A TEXT BOOK OF NEUROLOGY.

DR. A. CANNON AND DR. E. D. TRANCHILL HAYES are the conjoint authors of this book entitled "The Principles and Practice of Neurology";<sup>1</sup> and it is issued as a companion to "The Principles and Practice of Psychiatry", written by the same authors and published a year or so ago. The preface tells that the book is designed to be of special use to candidates for the diploma of psychological medicine and the M.D. degree in neurology.

Part I, covering fifty pages, is devoted to methods of clinical examination and has been provided by Professor Monrad-Krohn. It will be recognized as an abbreviated replica of a manual by the same writer, three editions of which have been reviewed in this journal.

Part II, the principal part of the book, covering 250 pages, is written by Dr. Cannon and Dr. Hayes, and is descriptive of nervous disease and disorder. The descriptions are brief, the field covered is comprehensive and conventional lines are followed, inasmuch as ætiology, pathology, symptomatology, differential diagnosis, prognosis and treatment are consecutively taken. These are worthy features. On examining the text, however, we notice many statements which, to say the least, are not in accord with accepted neurological doctrine. For example, "trigeminal neuralgia begins during adolescence as a rule and may soon disappear" is the reverse of the truth; "in some cases

of Ménière's disease removal of wax suffices to check the attacks" proclaims that in such cases the diagnosis must be at fault; and "aphasia is termed the loss of the visual and auditory memory of words" is the sort of definition which drew from Hughlings Jackson the ironical remark that we might as well speak of loss of memory of movements in a case of hemiplegia.

We are also tripped by frequent instances of careless writing, thus: "the prognosis is good if treatment is (be) conscientiously carried out" (we trust this does not refer to the physician); "a certain number of cases develop for the first time" (the disorder may first appear); on page 217 "These may be removed by operation" is a floating, contextless sentence; and the legend below figure 106 is quite unintelligible.

We notice further that the arrangement of subject matter calls for amendment, for instance, Schilder's disease, cerebral diplegia and injuries of the meninges are misplaced under the heading epidemic diseases of the nervous system, while hysteria is inadvertently tacked on to diseases of the spinal cord.

The publishers have performed their part most creditably, the print and paper being excellent and the illustrations (most of them original) clearly reproduced.

#### PROGRESS IN OPHTHALMOLOGY.

"RECENT ADVANCES IN OPHTHALMOLOGY", by Sir Stewart Duke-Elder, third edition, is what it claims to be, and is not merely a revision of previous editions.<sup>1</sup> It is well written on good paper and divided into two parts: "Physiology" and "Diseases of the Eye".

The section on physiology contains five chapters: "Vascular Circulation", "Intraocular Fluids", "Vitreous Body", "Intraocular Pressure", "The Effects of Drugs". It is a very concise but readable summary of the author's views on a large amount of practical work, much of which has been done by himself.

The second part contains a number of practical points which are undoubtedly useful. It is divided into seven chapters. The chapter on the cornea includes keratoplasty and it seems to give a gleam of hope that some day it may become a part of practical surgery. A number of methods of operation for detachment of the retina is described. Several refinements in cataract operation are described. Glaucoma is discussed from its various aspects, also its diagnosis and treatment.

Every ophthalmologist who reads the book will find it interesting and instructive from both a scientific and practical point of view.

#### A POPULAR BOOK ON THE CIRCULATORY SYSTEM.

"THE CIRCULATION OF THE BLOOD", by Winifred Parsons, M.A. (Cantab.), aims at giving a simple and up-to-date account of one of the most important of our bodily functions.<sup>2</sup> In this aim the author may be said to have succeeded. The book is written mainly for the interested general reader, but it also contains much that would be of value to nurses, medical students and also medical practitioners. A brief survey is given of the evolution of the circulatory system, starting from the simple amœba and working up through the different members of the animal kingdom.

Quite a good and up-to-date account is given of the structure, functions and operations of the different parts of the circulatory system and, although written in a semi-popular strain, it contains most of the important facts about the anatomy and physiology of this system.

In the last chapter a brief but interesting historical survey is presented.

<sup>1</sup> "The Principles and Practice of Neurology", by A. Cannon, K.C.A., M.D., Ph.D., D.P.M. (R.C.P. and S. London), and E. D. T. Hayes, M.D., D.P.M. (R.C.P. and S. London), with a Special Clinical Examination by G. H. Monrad-Krohn, M.D., F.R.C.P.; 1934. London: William Heinemann (Medical Books) Limited. Crown 4to., pp. 353, with illustrations. Price: 25s. net.

<sup>2</sup> "Recent Advances in Ophthalmology", by Sir Stewart Duke-Elder, M.A., D.Sc., Ph.D., M.D., Ch.B., F.R.C.S.; Third Edition; 1934. London: J. and A. Churchill, Limited. Pp. 430, with illustrations. Price: 15s.

<sup>3</sup> "The Circulation of the Blood", by W. Parsons, M.A.; 1934. London: The Sheldon Press. Crown 8vo., pp. 204, with illustrations. Price: 6s. net.

## The Medical Journal of Australia

SATURDAY, FEBRUARY 16, 1935.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

### THE MELBOURNE MEETING.

THE annual meeting of the British Medical Association to be held in Melbourne next September will be a unique event in the history of Australian medicine. Australian practitioners are used to congresses and similar gatherings; they make preparations many months ahead and generally manage to attend in considerable numbers. The September meeting, however, will be of much greater significance than an Australian medical congress can ever have. The British Medical Association has members in every part of the Empire, and the meeting thus becomes an Empire-wide gathering. Recently the Association held its annual meeting in Canada. Canada, as most Australian members know, has its own association, and this association is affiliated with the British Medical Association in England. In September next the Parent Body will be holding its annual meeting in the home of one of its overseas Branches. That the Victorian Branch is fortunate enough to have the honour of acting as hosts for the meeting is due

to the centenary celebrations of the City of Melbourne. It was originally hoped that the British Medical Association meeting would take place in Melbourne in 1934, but this could not be arranged. The Victorian Branch is naturally proud of its honour, but its members are most anxious that the other Australian Branches shall take an active share in the meeting and also in its preliminary arrangements. To this end two members from each Branch have been appointed to the Executive Committee. The Branches are thus kept informed of the progress made in the arrangements for the meeting and are able, through their representatives, to make suggestions. The first point that we would make is that the September meeting is a meeting which every Australian Branch should regard as one of its own activities and in which every Australian member should take a personal and practical interest.

In the second place attention should be drawn to the wide range covered by the sections. The names of the fourteen sections, with their office-bearers, were published in this journal on August 4, 1934. In every instance the president of the section has been chosen from Great Britain. Australian practitioners will thus have the privilege of coming into contact with members from the Old Country who are acknowledged to be leaders in the different branches of medical science. The scientific sessions of the meeting will be its main activity. These sessions, apart from their value for those who attend, will be a strong witness to the general public of the object for which the British Medical Association continually strives. Those of the community who look on the Association as something like a trades union will see a large delegation of medical men and women travelling across the world to meet their overseas colleagues in discussions on the cause of disease, its prevention and its treatment. This alone will make the effort of the organizers worth while.

With such a wonderful opportunity at hand, members of the Association in Australia will surely make every effort to attend the meeting next September. This is a meeting of the Association that every member is entitled to attend; no membership fee is payable. That attending members will

reap a personal benefit is undoubted, not only in the discussions that they will hear, but in the personal contact and exchange of views in the intervals between the sectional meetings. The President-Elect, Sir Richard Stawell, the General Secretary, Dr. J. P. Major, and the Executive Committee are not sparing themselves in their efforts to make the gathering successful. Of all the medical gatherings that have taken place in the Commonwealth of Australia this will be the most important and the most worth while. Such an opportunity may not arise again for many years. Medical practitioners who wish to leave their practices as a rule require to make their plans in advance. Now is the time to make the decision.

### Current Comment.

#### ADDICTION TO THE OPIUM ALKALOIDS.

It is a melancholy fact that recent literature concerning drug addiction has attained vast proportions and is rapidly increasing. Legislators throughout the world are diligently seeking methods to control the addiction menace, with only limited success. Those who engage in illicit drug traffic reap so great a financial harvest that they can view with equanimity the possibility of detection and punishment. Possibly in some instances complaisant authorities, with an eye to revenue, deliberately sanction such traffic. A problem which is seriously engaging pharmacologists is to find some substitute or compound of morphine which will have the same therapeutic value without its sinister possibilities, just as procaine (ethocaine or "Novocain"), which is not addictable, may replace the habit-forming cocaine. Cooperative research is being undertaken in the United States of America to try to find some such drug.

C. W. Edmunds, N. B. Eddy and L. F. Small have published their researches regarding morphine—its chemical construction in relation to its various actions, the importance of the phenanthrene nucleus, of the side-chains attached to the nucleus, and of the part each plays in the drug's action.<sup>1</sup> The morphine molecule was broken down and a morphine-like compound was built up, a start being made with the phenanthrene nucleus and side-chains being added. Observations were made on animals as to whether they were depressed or excited, the effects on respiration and the gastro-intestinal tract, the emetic action, and any analgesic effect. Addiction properties are not easily determined in animals, but the problem is being studied in dogs

and monkeys. Of importance is the comparative study of pairs or groups of compounds, differing from each other in one respect only—usually a substitution or change in one side-chain attached to the phenanthrene nucleus. Both morphine and codeine consist of the central phenanthrene nucleus with an oxygen bridge, a nitrogen-containing ring, hydroxyl groups and hydrogen atoms attached. A methyl group in codeine replaces the hydrogen of one of the hydroxyls in morphine. In codeine the phenolic hydroxyl of morphine is inactivated by the methyl group. Codeine is less depressant, less analgesic, and has less effect on respiration and the intestine than morphine, but is more convulsant. Fifteen pairs of derivatives allied to morphine were prepared, each differing from its sister compound, just as codeine differs from morphine, and in each case the same rule held. It was concluded that the presence of the free phenolic hydroxyl was important for the depressant and analgesic actions of morphine and for its respiratory effects. The second hydroxyl of morphine differs from the first, being alcoholic rather than phenolic. The ring to which the alcoholic hydroxyl is attached is aliphatic, the phenolic being connected to an aromatic ring. The alcoholic hydroxyl can be muzzled by methylation, acetylation or halogen substitution. A study of pairs of such compounds showed increased activity in all, especially as to analgesic and respiratory effects. The alcoholic hydroxyl in the 6-position seems to inhibit these actions of morphine. If its activity be suppressed, these effects are enhanced. Methylation of the phenolic hydroxyl decreases analgesic, depressant, respiratory and intestinal effects, and methylation of the alcoholic hydroxyl increases such actions above those exhibited by morphine. But muzzling either hydroxyl increases convulsant action and decreases emetic effect. Any chemical modification of the hydroxyl group in compounds of the morphine series decreases emetic action. Increased vomiting results when an additional free hydroxyl group is introduced. Phenanthrene itself is relatively inactive, being only a mild hypnotic, but when an hydroxyl group is inserted in the 3-position (as in the case of the phenolic hydroxyl in morphine) an analgesic effect appears and depressant action increases. If a second hydroxyl be inserted in the phenanthrene nucleus, these actions are further enhanced, as well as emetic and toxic effects.

The alcoholic hydroxyl in morphine can be removed and replaced by hydrogen or oxygen. In the first instance compounds are formed, termed desoxymorphines, of which dihydro-desoxymorphine is very active. This has no emetic effect and is only three times as toxic as morphine, but has an analgesic effect ten times and a depressant effect thirty to forty times as great as that of morphine. Replacement of the alcoholic hydroxyl by oxygen gives rise to compounds of the type of "Dilaudid" and "Dicodid", which are in clinical use. Many other compounds were investigated by Edmunds and his co-workers. It was found that the nitrogen-

<sup>1</sup> *The Journal of the American Medical Association*, November 10, 1934.

containing ring was of vital importance, and any modification of this ring, such as opening the ring and transforming it into an open chain, resulted in great loss of the morphine effects. It was not proved that the phenanthrene nucleus was the essential part of the morphine molecule. The synthetic work therefore extended to include diphenylene oxide, and compounds were made with it as a base. In building up such bodies many of the same groups were added as in the phenanthrene series. None of these displayed typical morphine action, but were more toxic and often exerted a greater analgesic effect. These researches show the correlation of chemical structure to pharmacological activity, but so far have not yielded an efficient substitute for morphine.

It has generally been assumed that codeine is non-addictable, although rare instances of such addiction have been recorded. C. K. Himmelsbach made an investigation of seven male morphine addicts who were stabilized on four daily injections of morphine over a period of ten to thirty days.<sup>1</sup> Then codeine acid tartrate was gradually substituted and stability maintained by codeine for eight to fourteen days. After abrupt and complete withdrawal of the codeine, mild evidence of abstinence appeared, which increased in severity until the fourth or fifth day, when recovery ensued. The picture differed from that of sudden morphine deprivation only in regard to the delayed onset of severe abstinence manifestations. Himmelsbach considers that codeine possesses definite addiction liability, but, being more expensive (in view of the larger doses necessary) and being less soluble, is not so likely to be used for addiction purposes. But the matter of solubility is over-stressed, as codeine phosphate is soluble in water, one in four, and clinical observation has shown, particularly in the quondam treatment of diabetes by codeine in large doses, that addiction possibility is so remote as to be practically negligible.

C. M. Stroud records the use of "Dilaudid" in the pain of cancer.<sup>1</sup> "Dilaudid" is didyromorphine hydrochloride, and at first was believed to be non-addictable. Stroud found that euphoria was rarely seen with this drug and that many patients who no longer needed it gave it up without any craving. Some patients preferred it to morphine. Only occasionally was craving noticed. Deterioration of character was less pronounced than with morphine, and morale at the approach of death was vastly better. Stroud considers "Dilaudid" an efficient analgesic in the control of constant pain, and he believes it less habit-forming than morphine. Unfortunately, in the same issue of the journal,<sup>1</sup> a letter from Paris states that Dr. Sainton found that "Dilaudid" gave the same euphoria and induced the same craving for larger and larger doses until a habit was formed, just as in the case of morphine and heroin. Stroud is unduly optimistic. It has been found also that "Eukodal" (di-hydro-oxy-codeinone hydrochloride) induces an especially per-

nicious habit of addiction. A similar preparation is "Acedicone", also with sinister possibilities. Curiously enough, "Dicodid" (di-hydro-codeinone) does not produce euphoria, and addiction is rare. To sum up, any drug giving euphoria possesses also the liability to engender addiction, and the aim of the pharmacologist must be to find some drug which will relieve pain and induce sleep without causing a state of euphoria.

#### ACTINOMYCOSIS OF THE FEMALE GENITAL ORGANS.

A MOST important contribution to medical literature has been made by S. S. Gardiner, of Newcastle, New South Wales. It has been published in the January, 1935, issue of *The Australian and New Zealand Journal of Surgery*. Gardiner has discussed actinomycosis of the Fallopian tubes. He has reported in detail a case of his own and has presented an extensive review of the literature. This is the first of a series of articles on actinomycosis that will be published by the same author. It is commonly believed that actinomycosis is a rare condition, but Gardiner shows that this is not so. During the last twenty years he has observed in private and hospital practice forty-six cases. In twenty-six instances the infection was located in the cervico-facial region, and in at least one of these the infection was due to a strictly aerobic organism. Nine cases involved the cutaneous and subcutaneous tissues. In four the infection was chiefly or entirely confined to the thoracic region; one of the four was apparently a case of primary catarrhal pulmonary actinomycosis. In four instances the infection was mainly confined to abdominal organs other than the genital organs. In two cases the urinary system was involved; one of these appeared to be a case of bilateral "excretory" actinomycosis and the other an example of ascending infection of the right kidney from a focus in the urethra. In one case the infection appeared to be an isolated infection of both Fallopian tubes; it is this case that has been discussed in the present instance. The clinical history is given in detail, and Professor D. A. Welsh, of the University of Sydney, has contributed a section on the results of his cytological and histological examinations. Gardiner states that the study of his cases has led him to the conclusions that are in conflict with some of the generally accepted opinions regarding actinomycosis. He holds: (i) that the disease is always a suppurative process, (ii) that actinomycosis is due to one distinct microorganism, (iii) that infection does not spread by lymphatic vessels. This is a study that demands careful consideration by all medical practitioners. Gardiner has gone to an immense amount of trouble in securing translations of articles from every country. He has made his research available to any medical practitioner interested in the subject, for a complete précis of the literature has been placed in the library of the Royal Australasian College of Surgeons in Melbourne.

<sup>1</sup> *The Journal of the American Medical Association*, November 10, 1934.

## Abstracts from Current Medical Literature.

### PHYSIOLOGY.

#### Splanchnic Pain.

In an attempt to throw additional light on the baffling problem of splanchnic pain, Edward A. Boyden and Leo G. Righer (*Journal of Clinical Investigation*, November, 1934) record a series of observations which were originally by-products of a group of experiments designed to test whether or not the human gall-bladder is subject to inhibitory reflexes originating in the gastro-intestinal tract. These experiments were subsequently repeated and elaborated in the belief that pain originating from ring contraction of the gut might be more specifically localized than sensations arising from inflammation or distension. The method of investigation consisted in sending an induction current through a Rehfuß tube, the metal end of which had been converted into an electrode. The second electrode was applied to the arm or leg. The subjects were eleven medical students, and the strength of current employed was similar to that used in ordinary physiological experiments. The maximum stimulus used was of a strength which was unbearable when applied to the lips but still tolerated by the gut. The effect of the current upon the stomach, as observed under the fluoroscope, was to induce a sphincteric contraction of the gut and then an increased peristalsis distal to the point of stimulation. This contraction was usually accompanied by some degree of abdominal rigidity. The sensation accompanying contraction of the gut ranged from barely perceptible feelings of pressure, gnawing sensations and heartburn to dull and severe colicky pain. Localization of these sensations was characterized by two general features: (i) the depth of the sensation (it seeming to come from well beneath the abdominal wall), (ii) the definiteness with which it could be located in the upper quadrants of the abdomen (the subject always pointing to the spot with one finger). The authors believe that the current used was not strong enough to penetrate the hollow viscus and still stimulate nerves in the anterior parietal peritoneum. They found, furthermore, that deep manual pressure over the lower end of the duodenum did not increase the pain, as it should have done if the pain was peritoneal in origin. In no case did maximum muscle tension cause the pain to be referred to the sides or back of the trunk. The latter phenomenon, when observed clinically, must therefore be due to extension of the lesion into the mesenteries or retroperitoneal tissues. When the

electrode was kept in one segment of the gut, but the body posture was changed, the site of pain usually shifted with it. When an area of the skin to which the patient had pointed was anesthetized, the pain migrated to a position outside the area. From these experiments the authors deduce that localization of visceral pain arising from spastic contraction of the gut is a viscerocutaneous radiation due to splanchnic bombardment of somatic neurones. At the same time that the pain migrated from an analgesic area it continued to be felt under that area. This suggests that visceral pain is normally an integration of impulses from both splanchnic and cutaneous sources and explains why such pain tends to follow the course of the gut. Confirmation of this theory awaits experiments with patients in whom the thoracic nerves have been cut.

#### Lethal Reduction of Temperature.

WHAT constitutes a lethal reduction of temperature on exposure to cold is a question of considerable theoretical and practical interest. In order to investigate it, F. K. Jackson and A. Alonge (*American Journal of Physiology*, September, 1934) have exposed a series of rabbits to varying temperatures by immersion in metal tubs filled with running water. Upon removal from the bath the rabbits were drained somewhat by squeezing of the fur. Their temperatures were taken *per rectum* and they were subjected to outdoor exposure on a window ledge for an average time of about seventeen minutes. (These experiments were conducted during winter.) At the end of this time their temperature was again noted, they were rubbed dry and placed by a warm radiator, where they had access to food and water. The results strongly indicate that there is no definite temperature above which life is always preserved and below which death always results. The reason for the variation is to be explained as due to other bodily factors which tend to maintain, to conserve and to return to normal the body temperature. Sixty-two per centum of the rabbits whose body temperature had been reduced to 19° C. died; 15.5% of those whose body temperature had been reduced to 19° to 25° C. died; and death came to only 2.7% of those whose body temperature had been reduced to only 25° C. or above.

#### Intraperitoneal Injection of Hypnotic Drugs.

THE peritoneal route of administration is utilized widely in the laboratory and clinic, especially for supplying fluids in dehydration. The extensive intraperitoneal use of hypnotics by veterinary surgeons is important, and more recently this route has been used for hypnosis in man. Gene H. Kistler (*Journal of Laboratory and Clinical Medicine*,

November, 1934) considers that intraperitoneal injection is not without its dangers. In order to collect data on the subject he has examined the stomach and bowel of approximately 150 dogs and 100 cats after periods of from ten minutes to three months following routine intraperitoneal injection of hypnotics for anaesthesia. Six accidental injuries to the bowel were found. One animal died of acute generalized peritonitis, the result of perforation. This author considers that the bowel is punctured or intraintestinal injection is made more frequently during intraperitoneal administration than is generally supposed. Simple needle puncture wounds of the stomach and small bowel heal without consequence, but the barbituric acid hypnotics, when injected into the wall of the stomach and small bowel, rapidly produce necrosis of the lining and subsequent changes of the deeper tissues. These changes are probably due to alkalinity and slow absorption of the substance. Although there is distinct tendency for the lesions to heal, perforation of the wall may occur. Glucose and sodium chloride solutions, unless concentrated, produce no damage when small amounts are injected into the wall of the stomach or small bowel. The author does not suggest that intraperitoneal administration should be discouraged, but that precautions should be taken to insure peritoneal absorption and freedom from injury to the gastro-intestinal tract. Particular care should be exercised if the stomach or bowel is distended.

#### The Velocity of the Blood Flow.

OWING to the technical difficulties in the continuous measurement of blood flow in man, our knowledge of the changes produced in blood flow through the extremities by exercises and changes in posture remains incomplete. Gibbs has recently described a thermo-electric blood flow recorder in the form of a needle. His method requires nothing more than a venipuncture and is therefore adapted for use on human subjects. S. H. Procter and L. Dexter (*American Journal of Physiology*, October 1, 1934) have found Gibbs's method useful and practicable as a means for measuring continuously the relative velocity of blood flow and, in certain instances, qualitative changes in volume flow. Where it was desired to record changes in velocity in a vessel which was visible, a solid needle in which hot and cold thermo-junctions had been incorporated had certain advantages over an open needle into which the thin wires containing the junctions were threaded after venipuncture. These experiments showed that the velocities of blood flow in the superficial and deep veins of the arm behaved antagonistically with exercise of that arm, the flow in the deeper veins becoming more rapid and in the superficial veins slower than at rest. With moderately severe exercise

involving chiefly the lower extremities (pedalling on a stationary bicycle) there was no change in the blood flow in the veins of the upper extremities. In the superficial and deep veins of the upper extremities the velocity of blood flow was slower with the arm held in the erect position, and more rapid with the arm hanging down, than when the arm is in the horizontal position.

## BIOLOGICAL CHEMISTRY.

### Intercellular Substance and Ascorbic Acid (Vitamin C).

V. MENKIN, BURT WOLBACH AND M. MENKIN (*American Journal of Pathology*, September, 1934) have found that ascorbic acid (vitamin C in crystalline form) administered orally or parenterally to scorbutic guinea-pigs induces reparative processes, as demonstrated by the renewal of dentine formation in the incisor teeth and by the deposition of osteoid matrix and chondromucin at the costo-chondral junction. These results on the formation of intercellular substance are in accord with those previously obtained by Wolbach and Howe by feeding orange juice. They furnish additional support for the view that ascorbic acid is indistinguishable from vitamin C. The evidence obtained indicates that a relatively simple chemical substance, ascorbic acid, controls the deposition of intercellular substance. The possible mechanism involved in this reaction is discussed from the standpoint of the properties of ascorbic acid as a reducing agent in relation to cellular oxidations.

### Cereals and Rickets.

H. M. BRUCE AND R. K. CALLOW (*Biochemical Journal*, Volume XXVIII, Number 2, 1934) have attempted to elucidate the phenomenon noted by Mellanby and others, that certain cereals (oats, maize *et cetera*) have an anticalcifying factor which has the effect of producing or intensifying rickets in dogs or rats, and of retarding the calcification of the teeth in dogs. This could not be explained by the absolute or relative amounts of calcium or phosphorus in the cereals. Most cereals are rich in phosphorus, but from 50% to 80% of this may be present in the form of inositolphosphoric acid (phytin). Phosphorus in this combination is probably not available to the body. The diets used had a high calcium/phosphorus ratio. That the diets were lacking only in available phosphorus was indicated by the large increase in antirachitic potency on addition of sodium phosphate alone. Addition to the basal rachitogenic diet of "Vitaphos" (calcium-magnesium-inositolphosphoric acid) caused a negligible rise in the antirachitic

potency of the diet. The efficiency of oatmeal as an antirachitic factor is intermediate between sodium phosphate and "Vitaphos". When the oatmeal was treated with dilute hydrochloric acid the inositolphosphoric acid was hydrolysed, the phosphorus being thus rendered available and the antirachitic potency of the oatmeal being thereby raised to the same extent as by the addition of an equivalent amount of sodium phosphate. There was evidence that the effect of treatment with hydrochloric acid was due to the destruction of a rachitogenic factor in the cereal. The differences in the antirachitic potency of various cereals can be explained by the differences in the content of phosphorus in available forms and in the unavailable form of inositolphosphoric acid. The analysis of the phosphorus content of the food may therefore be misleading if the availability of the phosphorus is not taken into account. When the deficiency of calcium is the factor limiting calcification, the cereals have a rachitogenic effect, because the inositolphosphoric acid forms insoluble calcium salts, and this intensifies the deficiency of calcium, in a manner comparable with the effect of excess phosphoric acid. Beside this anticalcifying "toxamin", cereals are thought to contain toxins responsible for the production of nervous symptoms due to degenerations in the central nervous system. Prolonged feeding of inositolphosphoric acid to monkeys failed to produce any nervous symptoms, thus indicating that this substance is not responsible for the nervous degenerations noted by Mellanby and by Stockman and Johnson.

### Physiological Action of Vitamin C.

VICTOR DEMOLE (*Biochemical Journal*, Volume XXVIII, Number 3, 1934) conducted experiments to determine whether the administration of large doses of ascorbic acid (vitamin C) would produce hypervitaminosis, as in the case of vitamins A and D. Various animals were used, and doses of up to five grammes per kilogram were administered. In all cases the doses were well tolerated and no symptoms of hypervitaminosis were observed. No histological changes were seen in the kidneys, livers, hearts or lungs. After administration of 0.1 gramme per kilogram subcutaneously to a dog 75% was excreted in the urine during the next nine hours. The complete identity in biological action of synthetic l-ascorbic acid with the pure crystalline vitamin C from natural sources was established.

### Hæmoglobin Regeneration.

W. C. SHERMAN, C. A. ELVEHJEM AND B. E. HART (*Journal of Biological Chemistry*, October, 1934) have studied the value of egg yolk as a source of iron and copper for hæmoglobin

regeneration. Estimation of iron by Hill's dipyrldyl method showed that nearly 100% is available. The availability of the iron was not affected by boiling or by extraction with ether. Although egg yolk is a rich source of available iron, it was found that hæmoglobin regeneration in anæmic rats was very slow and incomplete when egg yolk was used as a supplement, either alone or with small amounts of added copper (in the form of copper sulphate in doses of 0.05 milligramme daily). That the iron was available was indicated by the fact that considerable amounts of iron were stored in the treated rats. It was further found that when larger amounts of copper were supplied in the form of copper sulphate (1.00 milligramme daily) in addition to the egg yolk, regeneration was rapid and complete. It is suggested that the availability of the copper is reduced by the formation of copper sulphide, which is insoluble and unavailable.

### Pernicious Anæmia.

LOUIS KLEIN AND JOHN FREDERICK WILKINSON have conducted researches to elucidate the nature of hæmopoietin—the antianæmic substance present in hog's stomach (*Biochemical Journal*, Volume XXVIII, Number 5, 1934). This substance is precipitated with proteins, gives all the protein reactions, and is soluble in water, but not in 70% alcohol. It has not been possible to separate proteins from the action fraction without complete loss of hæmopoietic activity. Fractions have been prepared from hog's stomach with but little peptic activity, but still hæmopoietically active. Hæmopoietin loses its antianæmic property on heating at 60° to 65° C. for half an hour. When this thermolabile hæmopoietin was incubated with beef muscle a hæmopoietically active material was obtained. This substance was relatively thermostable and closely resembled the active principle obtained from liver. It can be freed from proteins and prepared in a form suitable for intramuscular injection by a technique similar to that used for obtaining from liver fractions suitable for parenteral use. Pepsin itself, or fractions from the hog's stomach shown clinically to be free from hæmopoietin, gave negative results when incubated with beef muscle. The authors consider that the relationship between the antianæmic principles in stomach and liver is that of an enzyme to an end-product. The enzyme hæmopoietin is present in hog's stomach and normal gastric juice. The substrate is some unknown substance in beef muscle, and the result of the enzymic activity is a thermostable, hæmopoietically active substance which is absorbed and stored in the liver until required by the body for the production of red blood corpuscles. All the preparations were tested for hæmopoietic activity by clinical trial on pernicious anæmia patients.

## Special Article.

### ANNUAL MEETING OF THE BRITISH MEDICAL ASSOCIATION, MELBOURNE, 1935.

THE following article on Melbourne has been written by Dr. Felix Meyer at the request of a subcommittee of the executive committee in connexion with the annual meeting of the British Medical Association, Melbourne, 1935.

#### EARLY HISTORY OF MELBOURNE.

"This will be the place for a village." Thus John Batman in his diary on June 8, 1835—a phrase that has passed into history. Batman, a native of New South Wales, had crossed over from Tasmania, then Van Diemen's Land, as a representative of a syndicate of speculators who wished to settle in Victoria. He had come by boat "about six miles up the river" (the Yarra) from its junction with the Saltwater River, and "found the river all good water and very deep". His actual landing place on the north bank of the Yarra is marked by a stone set in the pavement at the intersection of Flinders and William Streets, a cable's length from what was known as Batman's Hill, which was cut down later to provide the present site of the Spencer Street Railway Station. Sixty years before that landing, on April 20, 1770, Captain James Cook, in the *Endeavour*, had sighted the coast of Victoria some ten miles south of Cape Everard and had named Cape Howe. On January 4, 1798, George Bass, an assistant naval surgeon, after a five weeks' voyage from Sydney in a whaleboat, landed at Western Port, a large inlet on the south coast of Victoria. Some escaped convicts and later some of the shipwrecked crew of the *Sydney Cove* had previously found landing on this part of the coast. But if Bass was not one of the first white men to touch the shores of the future State of Victoria, he certainly was the first medical man to do so. And it was another medical man, Dr. McCallum, surgeon of the fifty ton schooner *Cumberland*, the third vessel to enter Port Phillip Heads, who was one of the first white men to visit the site of Melbourne. He was a member of the expedition with Charles Grimes, Acting Surveyor-General, who had been sent from Sydney in the *Cumberland*, commanded by Lieutenant Charles Robbins, to survey Port Phillip, which had been discovered by Murray in the brig *Lady Nelson* on January 5, 1802. In his "Story of Melbourne" Mr. Alfred S. Kenyon says:

On the 2nd February they landed between St. Kilda and Port Melbourne and came to a large river; the ground was swampy on one side and high on the other. This was the Yarra Yarra near its mouth. On the 3rd, Thursday, Robbins, Grimes, and Flemming, with five sailors, rowed up the Yarra as far as the junction of the Saltwater, then up the Saltwater River, now the Maribyrnong, as far as Solomon's Ford, Keilor. Returning next morning, they continued up the Yarra, and found excellent water after a few miles. Landing at Batman's Hill, they had dinner, and then examined the river for a few miles further. Thus on the 4th February, 1803, Melbourne was first visited by white men.

With the discovery of Dight's Falls the journey ended. The Freshwater River (Yarra) and the quality of the land appealed to Flemming; this land, he says in his report, "is the most eligible place for a settlement". Batman having obtained possession of 600,000 acres from the aborigines by the payment of £200 and a collection of articles, some useful, some ornamental, and a "contract" later disallowed by the Government, returned to Tasmania. In his absence John Pascoe Fawkner, of Launceston, a Jack of all trades, shop-keeper, farmer, hotel-keeper, editor of the *Launceston Advertiser*, came across with a party and found his way to the site selected by Batman, built a little hut and actually turned the first sod of the future city on August 30, 1835. When Batman returned on November 9 there were, naturally, differences between the

rival pioneers as to the division of lands; these were finally satisfactorily adjusted. A point interesting to the medical profession is that with Batman on this occasion came Melbourne's first doctor, Dr. Barry Cotter, L.F.P.S.G. It is on the extract from Batman's journal, quoted at the beginning of this article, that is based the claim, which has been so hotly contested by Fawkner and his supporters, that Batman founded Melbourne.

From such origins here briefly outlined, the settlement grew rapidly. In 1837 Governor Bourke visited it and named it Melbourne, and on June 1, the same year, was held Melbourne's first great historic land sale. The year 1851 saw the separation of Port Phillip from New South Wales. It became a colony with the name of Victoria; and in the same year the discovery of gold in several places led to a great influx of immigration. In 1854 Sir Charles Hotham was appointed the first Governor of the State; the first railway in Australia was opened from Melbourne to Sandridge (Port Melbourne), and the first telegraph line in Australia, between Melbourne and Williamstown, was erected. With the granting of a constitution to Victoria and the opening of its first parliament in 1856, the city developed an increasing civic sense and a stabilization of the social life of the community. Hence onward the history of Melbourne is one of peaceful progress in the best sense of the term. True, there have been ups and downs, one small rebellion, strikes, the land boom and other disabilities. Today Melbourne is the sixth city of the Empire, with an urban population of 1,028,000 or, with its outlying suburbs, Greater Melbourne, nearly a million and a half.

After a hundred years we may say with the Roman poet: "Here where now there is a city, was formerly nothing but the site of a city."

#### PRESENT-DAY MELBOURNE.

The visitor to Melbourne will be at once impressed with the fine wide and well paved streets running at right angles, and the handsome public buildings, attractive in their strength and dignity. Speaking generally, the architecture of the city does not conform to a particular type—there is no uniformity of style—and while some are, by way of contrast with others, of outstanding height, structures of thirty or forty stories—the skyscrapers of American cities—are not as yet in evidence. Especially will the visitor be impressed with the beautiful boulevards that lead from the city to the suburbs, and the wide expanse of public parks and gardens, which occupy a fourth of its area. Melbourne is in truth a garden-city. To fly over it is to realize the generous open spaces of the refreshingly green parklands that touch the boundaries of the business part of the city, and link it up with the suburban area; and as one circles over the suburbs, one looks down in every direction on parks and gardens, public and private. Truly the wise forefathers of the hamlet bequeathed a goodly heritage when they set aside in the original crown grants some 2,500 acres for public reserves in the city proper. Starting at the eastern end of Collins Street, one can walk all the way to St. Kilda at the seaside, a distance of four miles, through public gardens and parks, without stepping off green turf, except for the crossing of a few main roads. Melbourne still lacks a central square, and though many suggestions have been forthcoming to supply the want, no plan for the creation of a civic centre for inner Melbourne has so far been satisfactorily evolved.

As has been said, the lay-out of Melbourne is rectangular, the business section of the city forming a block about a mile and a half in length and a mile in breadth. The streets for the most part are broad, but there are numerous lanes. There are four main thoroughfares, Collins Street and Bourke Street running east and west and Elizabeth Street and Swanston Street intersecting them. To stand at the eastern end of Collins Street in the quiet of a Sunday morning, to follow its pleasing, unbroken course, with its imposing buildings, the two fine churches, the Scots' and the Independent, with their beauty of soaring spire and campanile, the tender green of planes and other deciduous trees which border the pavement on either side of the eastern end of the street, is to understand the pride of the citizens of Melbourne in their hundred years old city.

**Public Buildings.**

**The Town Hall.**—The Town Hall is at the north-east corner of Collins and Swanston Streets. It is in classic Renaissance style. An unusual feature is the portico of generous height and proportions, beneath which a carriage path allows of the passage of motor cars *et cetera* to the foot of the ascending steps to the foyer leading to the hall itself. Above the portico is a balcony looking on to the street, much favoured on public occasions connected with municipal ceremonials and an excellent point of view for processions and other displays. The auditorium will seat 2,500 persons; its walls are decorated with frescoes of classical subjects in an agreeable colour scheme. The magnificent organ is one of the world's greatest instruments. Free organ recitals are given throughout the greater part of the year. The city is very fortunate in

and opened to the public in 1856 in a room about fifty feet square, now the central portion of the Technological Museum. It is a huge structure housing the library, a notable art gallery, a natural history museum and a technological museum. It is classical in style, and the visitor, as he faces its western front, is at once impressed by its simple dignity and fine proportions. The main entrance is from Swanston Street, and on the terrace in front of the buildings are the statue of Sir Redmond Barry, sometime Chief Justice of Victoria, "erected by a grateful public" as a memorial for his many public services, particularly in the development of the institution; a bronze group of Saint George and the Dragon, by Sir J. E. Boehm, R.A.; and a bronze equestrian statue of Jeanne d'Arc, by E. Fremiet, a replica of the statue in the Place des Pyramides, Paris. On entering the building the visitor



Skyline of Melbourne from the River Yarra.

[By courtesy of *The Herald*.]

having a distinguished organist in Mr. William McKie. During the centenary weeks one of the most beautiful sights in the city was the rich blazonry of the heraldic banners and the coats of arms of former governors colourfully decorative in the sunlight and at night, flood-lit, an illumination of unusual splendour.

**The Manchester Unity Building.**—On the opposite corner to the Town Hall is the newly erected Manchester Unity Building, one of the tallest buildings in the city, graceful, with column-like effect. Its modern style, evidence of American influence, is in marked contrast to the prevailing type of architecture.

**The Public Library.**—Passing northwards along the east side of Swanston Street, beyond its busiest section, about one-third of a mile from the Town Hall, one comes to the State Public Library of Victoria, which was founded in 1853, mainly owing to the exertions of Sir Redmond Barry,

will find a number of marble statues and other exhibits, including an Egyptian mummy, in the vestibule, on each side of which are halls devoted to ethnology. There is a children's room on the left. One of the features of the building is the marble staircase, seven feet wide, branching on each side from the vestibule leading to the reference room and the technological museum. The vestibule in which it is contained is entirely lined with marble from Buchan, in Gippsland.

The present library building, which was opened on November 14, 1913, consists of a basement, a ground floor, a first floor and three galleries, and has accommodation for 2,000,000 books. The ground floor is occupied by the newspaper room, an octagonal chamber about 149 feet in diameter, containing more than 25,000 bound volumes. A very fine collection of Victorian newspapers is housed in this room, and here also may be found files of the

leading papers of Australia and representative papers from most countries of the world. Owing to lack of space in the picture galleries, many oil paintings are hung on the walls of this room.

The reading room on the first floor is an octagonal chamber, approximately 220 feet square. It is surrounded by an annulus about 14 feet wide. This annulus contains offices on the main floor and galleries containing stack-rooms for books on the upper floors. The surrounding galleries and museums shut off the noise of the city, while ample provision is made for ventilation and light by courtyards on four sides of the octagon. The dome of the great reading room, which is believed to be the largest ferro-concrete dome in the world, is 114 feet in diameter and 114 feet in height, and its floor space gives ample accommodation for 320 readers. It contains 3,800 square feet of double glass, the inner portion consisting of Luxfer prisms, which distribute the light equally and prevent

in existence, consists of about 12,000 specimens. The Spencer Hall of Australian Ethnology houses the valuable Australian ethnological and natural history collections presented to the museum by Sir Walter Baldwin Spencer. The culture of the aborigines is illustrated by 8,500 specimens.

The art collections occupy ten galleries, one of which is devoted to the works of Australian artists. In the way of endowment the National Art Gallery of Melbourne is the wealthiest in the Empire. The munificent bequest of Mr. Alfred Felton, a Melbourne merchant, who died in 1904 and left his estate in trust, to be equally divided between gifts to charities and the purchase of works of art for the National Gallery, has enabled the trustees of the gallery to enrich the collection from time to time with most valuable paintings and other works of art, including some very valuable old masters. Since 1904 approximately £400,000 have been spent from funds provided by the Felton



Parliament House, Melbourne.

[By courtesy of *The Australasian*.

glare. A complete system of heating and ventilation is provided. The total number of volumes in the library is over half a million. About 32,000 volumes are placed on the open shelves in this room, and printed guides are to be found on the tables. The library is specially well represented in the sections devoted to art, music, English literature, heraldry and genealogy, bibliography, Australiana, Shakesperiana, and history, and has a remarkable collection of examples of the productions of early printers and of modern private presses.

There is also a lending branch attached to the library, containing about 67,000 volumes. The technological museum, which occupies two halls, is excellently illustrative of the industrial arts. The section devoted to Australian and Victorian timbers is an interesting feature.

In the National Museum of Zoology, Geology and Ethnology the exhibited collections occupy five halls and two galleries, having a total area of well over one acre. They are rich in Australian zoological and palaeontological specimens; the Australian collection of fossils, the finest

Bequest. Other notable gifts from generously-minded citizens have increased the extent and importance of the National Gallery.

*The University.*—The University is in the northern part of the city, away from its din, and is reached by tram from its centre in a few minutes. Its foundation stone was laid in 1854. It is set in spacious grounds, which contain, in addition to the main building, the four affiliated colleges, Trinity, Ormond, Queen's and Newman, the professors' residences, the Training College, and the various departments of special science. If the distant prospect reveals no dreaming spires or antique towers, yet its eighty years have given a mellowed charm to the brown stone, and its shadowed cloisters have an air of ancientry. Through them one steps on to the quadrangle, in the season bright with red camellias. There are no "backs", no Isis or Cam, and the only "watery glade" is the artificial lake bordered by gently sloping lawns; but there is ample roaming ground for the hundreds of students, and a cafeteria. Besides the older building there are the

Conservatorium of Music with the Melba Hall, the School of Geology and the Arts Building, with its fine clock tower. On the north-east or Carlton side of the grounds is the Medical School, the first to be established in Australia, a large, compact group; the Department of Anatomy is said to be the largest in the Empire. The outstanding feature of the University is the Wilson Hall, a beautiful Gothic building, the gift of the late Sir Samuel Wilson, with its stained glass window presented by the late Mr. Edward Stevens. Its walls are decorated with portraits in oil of past chancellors and professors of the University. On Commencement Day, when the conferring of degrees takes place, the hall is taxed to its utmost capacity. Other ceremonies of the alma mater, social functions, concerts and special lectures, have their setting in this hall. The four affiliated colleges are distinctive in their architecture. Their generous proportions reflect the generosity of their benefactors.

**Parliament House.**—Situated in Spring Street, fronting the eastern end of Bourke Street, is Parliament House, which is a glorious example of Doric architecture. Standing on the rise, with its broad flights of steps leading up to the portico, and with its massive, beautifully proportioned columns, it impresses greatly. One gives it pride of place in Melbourne's fine buildings. From the vestibule one passes into the Queen's Hall, which is paved with a very fine mosaic. The two halls, that of the Legislative Council and the Legislative Chamber, are spacious and ornate—each is as large as the House of Commons. Their architecture is Græco-Roman. The panellings and vaulted ceilings are richly decorated and the furnishings luxurious and in excellent taste. The library, with a handsome dome, houses nearly 100,000 volumes. Sculptures by the late Sir Bertram MacKennal adorn the highest part of Parliament House on its southern wall.

**The Exhibition Building.**—The Exhibition Building, the largest public building in Melbourne, is situated in the north of the city, a short journey by tram. Standing on a rise, it dwarfs all surrounding structures; its dome can be seen from the upper reaches of the bay. Its immense hall, the largest in the city, is used for industrial exhibitions, musical festivals and the great gatherings of Armistice Day, Anniversary Day, Eight-Hour Day and other occasions. Its large oval is used for motor cycle races and other sports. An aquarium and war museum are part of its attraction, and its fine, well laid out gardens are very popular with the children of the adjacent suburbs of Fitzroy and Carlton.

**The Law Courts.**—The Law Courts occupy an elevated position at the corner of Lonsdale and William Streets.

The building, which has a conspicuous dome, is a solid block built on to the pavement. Simple in line, it appeals by its dignity.

**Other Public Buildings.**—A block further on the east side of William Street is the Royal Mint. A building which stands out, combining the artistic with the utilitarian, is the Metropolitan Fire Station at the corner of Victoria Street and Gisborne Street. It is built on an elevation of 140 feet above sea level, and from its look-out tower, 150 feet in height, commands a magnificent panorama. Collins Street is admittedly the city's finest street. Many imposing buildings line its sides throughout its whole length. Besides the two churches already mentioned at the

east end, the Temperance and General Building at the corner of Russell Street is a fine example of the newer type of architecture. The banks and the insurance offices, individual studies, are all handsome structures, their façades attractive with columns of beautifully polished granite, grey or red, and glistening facings of coloured stone from Victorian quarries. The eastern part of Collins Street was, twenty years ago, given up mainly to doctors and dentists. Today, shops, tea-rooms, pharmacies *et cetera* are ranged among the brass plates—the atmosphere is not quite the same. Residential flats have also come to stay. Besides the Melbourne Club, long established in this part, two of Melbourne's best hotels are here. A little way beyond the end of Collins Street is a very beautiful fountain, the work of a prisoner many years ago. For grace and poetry of design it has no equal in the city.

Space will not permit detailed accounts of many interesting features of the city. There are shops and warehouses covering many acres from Bourke to Lonsdale Streets worthy of any great city. Outstanding are the enormous Myer emporium in Bourke Street, the largest of its kind in the Southern

Hemisphere, *The Argus* and *The Australasian* offices, now in Elizabeth Street, for many years in Collins Street, the *Herald* office, in Flinders Street, all in commanding positions and easy of access.

At the corner of Russell Street and Victoria Street, on the site of what was for many years the Melbourne Gaol, stands now the Emily McPherson College of Domestic Economy, the munificent gift of Sir William and the late Lady McPherson.

#### The National War Memorial of Victoria.

The National War Memorial of Victoria, or Shrine of Remembrance, is erected to the memory of the men and women of the State who served the Empire in the Great War, abroad and at home, more especially those who made the supreme sacrifice. It occupies a commanding



The Exhibition Building, Melbourne.

[By courtesy of *The Star*.

site on a hill gently rising to 83 feet above sea level, on a parkland bordered on the southern and western aspects by Domain Road and St. Kilda Road and overlooking the Botanic Gardens and the grounds of Government House. With its crown 207 feet above sea level and its northern side directly facing Swanston Street, it is clearly visible from nearly every part of the city, dominating the landscape—a landmark for the shipping, seen far out in Port Phillip Bay. The spirit and the purpose of the memorial are there for all to understand in the inscriptions carved on the beautiful west and east stone walls:

**On the west wall:**

Let all men know that this is holy ground, this shrine established in the hearts of men as on the solid earth commemorates a people's fortitude and sacrifice. Ye therefore that come after give remembrance.

**On the east wall:**

This monument was erected by a grateful people to the honoured memory of the men and women of Victoria who served the Empire in the Great War of 1914-1918.

The memorial is designed after the mausoleum erected at Halicarnassus in 353 B.C. The walls on the east and west are plain, but those on the north and south have porticos supported by beautiful fluted Doric columns of granite, each column being twenty-one feet high. The groups of sculpture relate the tragedy of war, the triumphs of peace, the fine arts and science, and allegories of agriculture and pastoral industry symbolical of Australia.

Immediately within the external walls of the memorial a spacious ambulatory encompasses the inner shrine, from which four separate flights of stairs ascend to the high level exterior promenades and descend to the crypt. The ambulatory is provided with a spaced series of niches indented in the outer walls, in which are housed forty-two beautifully modelled caskets of bronze that contain the Books of Remembrance. The parchment pages of these books are inscribed with illuminated manuscript records of the names of all members of the Australian Imperial Force who were born or who enlisted in Victoria, who served overseas during the Great War or who died in camp prior to embarkation. Here also is the King's Book inscribed with the special message of His Majesty King George V: "Let their names be for ever held in proud remembrance." The true inwardness of the memorial is the inner shrine, a temple within a temple, a beautifully proportioned quadrilateral chamber. Here, sunk below the pavement, is a plain slab of dark marble, the Rock of Remembrance, inscribed: "Greater love hath no man." The grave beauty of this holy of holies is made more deeply impressive by an arrangement by which, through a special aperture in the roof, a ray of sunlight falls on the centre of the Rock of Remembrance at the eleventh hour of the eleventh day of the eleventh month in every year. Two high promenades surround the base of the dome, and from these parapets spectators get a splendid panoramic view of the city, the surrounding country, the distant mountain ranges and the bay. Returned soldiers guard the shrine in watches throughout the twenty-four hours of the day. Flood-lit at night, it is seen as a glowing pyramid of pure light, a consecration and a prayer.

**The St. Kilda Road.**

Melbourne's finest boulevard, of which it is justly proud, is the main artery to the city from the south, linking up with the esplanade at St. Kilda, the pier and beach, and with a foreshore road that follows the contour of the bay to Brighton, Beaumaris, and the farther seaside resorts—a picturesque route. Beginning at St. Kilda Junction, the St. Kilda Road runs between double rows of deciduous trees and flowering scarlet gums. These, set in strips of well kept lawns, make a triple division of the road, providing passage for trams and heavy traffic in the centre, and motor cars on either side. Both sides of the road for more than a mile are residential. On the east side are noticed the Deaf and Dumb Institute, the recently rebuilt Wesley College, a modern reconstruction, the

generous gift of Messrs. A. and G. Nicholas, and the Royal Victorian Institute for the Blind. Many fine houses and villas, with their gardens, line both sides of this section of the road. At the corner of Toorak Road the new Synagogue, with its imposing dome, meets the eye, and a little farther on is the Church of England Grammar School, an old world blue-stone building, mellowed by time, with an English School look of its own, set in grounds of ample space. Passing the Domain Road, the northern boundary of the Grammar School, one comes immediately to the Domain, with the National War Memorial, already described. On the opposite side is the South African Memorial. Further on is the entrance to Government House, with the bronze equestrian statue of the Marquis of Linlithgow, the first Governor-General of the Commonwealth. Opposite is the massive blue-stone building of the Victoria Barracks, begun in 1854, with two 42-pounders captured in the Crimea on either side of the doorway, and on the lawns in front a German and a Turkish howitzer captured by Australian troops in the Great War. After the Barracks come the Prince Henry Hospital (Homœopathic), the Police Barracks, the memorial to Nurse Edith Cavell, and the bronze statue of Robert Burns. On the east side are the Queen Victoria Gardens, with the Queen Victoria Memorial and the bronze equestrian statue of King Edward VII, the work of Sir Bertram MacKennal, a fine example of his art. And so along the tree-lined road to the gate of the city—Prince's Bridge. Facing the gate is St. Paul's Cathedral, with its recently completed spires. This is the busiest section of the city, being the main outlet for its traffic, with its two railway stations, one on either side of the city end of the bridge. Standing on the bridge, we look upstream to that part of the river associated with recreation—boat races, pleasure trips, swimming and diving exhibitions. Downstream we look towards the beginning of the wharves, to where the shipping traffic touches the heart of the city, where sea and land transport meet. Here, below Queen's Bridge, the river basin, above which the shipping cannot go, is the landing place of incoming and outgoing cargoes—the landing place of Batman a hundred years ago.

**Alexandra Avenue.**

This beautiful avenue branches off at an angle from Prince's Bridge and follows the south side of the Yarra—a double row of planes and poplars on the broad green sward on either side adds to its breadth as it passes through the Queen Victoria and Alexandra Gardens to the Yarra's green bank which it follows. On the right the Botanic Gardens add to its wealth of green shelter. It is a favourite drive to and from the city—a perfect road for motor cars. Running parallel with it are a foot walk and a tan track for equestrians. This part of the river, known as the Henley Course, is the mile of water for the annual public schools' eight-oared race and the Henley Regatta, and on these occasions the scene on both banks of the river is one of fascinating life and colour, the spectators numbering thousands. The picturesque houseboats, the gaily decorated canoes, the boating dress of men and maids in their infinite variety, make a colourful pageant, to which the cool green leafy avenue, with its flickering sunlight on the throngs who parade the green sward, forms an ideal setting for the picture which Melbourne paints for itself to make an artist's holiday. And on Henley night a display of fireworks gives to the scene a touch of fairyland.

**The Public Gardens.**

*The Botanic Gardens.*—By common consent Melbourne's finest public gardens were started in 1852 by Baron F. von Mueller, Government Botanist, a writer of many books, and explorer. He became Director in 1857, retiring in 1873, and it was his successor, W. R. Gillfoyle, who planned out the splendid system of the gardens as we know them today. The gardens are part of the great reserve of 320 acres lying on rising land immediately south of the city boundary formed by the Yarra. The western side of the reserve marches with the St. Kilda Road, Domain Road and Anderson Street in South Yarra being the southern and eastern boundaries. This reserve

comprises, besides the Botanic Gardens, the Domain, the Queen Victoria and the Alexandra Gardens. On the highest part of this reserve stands Government House with its grounds. The gardens can be reached by tram from the

being by a gate opening on a path leading up to the classical little Temple of the Winds, whence one can overlook the river and get a view of the city and its spires. The site for these gardens was a happy choice, for the



The Botanical Gardens, Melbourne.

[By courtesy of *The Herald*.]

city in a few minutes via St. Kilda Road, or by motor car, a shorter route, through the Domain, passing by the Shrine of Remembrance. A third route is by way of the Alexandra Avenue with the river alongside, the entrance

103 acres which they occupy, are an undulating hillside, falling gently to the river, which that master gardener, W. R. Guilfoyle, shaped to sloping lawns and glades, fern gully and palm grove, leading to an islanded lake, a

sanctuary for black swan, coots, wild duck and other waterfowl, which nest and find shelter in the sedge. Looking from the eastern lawn one's eye travels over lake and wooded upland to the stately tower of Government House "bosom'd high in tufted trees"; and on all sides is beauty of trees and lawn, flowers and shrubs of every description, trees massed naturally and grouped as far as may be in botanical order. Medicinal plants and herbs keep "discreetly" to themselves. Australian flora are to be found at the southern boundary of the gardens. Oaks from all parts of the world flourish together on the oak lawn. Calodendrons, Chinese pawlonias, of scent indescribable, jacarandas, coral and flame trees when cloaked with their blooms add colour, in mass, of mauves, blue, coral and flaming red—a natural impressionist picture. Tropical, temperate and northern vegetation finds a healthy habitat here—a single sequoia of moderate height is the sole representative of the giant Californians, and Himalayans are numerous. A sound knowledge of their requirements has made it possible to adapt the local conditions to aspects favourable to the growth of strangers to this land. Rhododendrons, azaleas, cinerarias, dahlias flourish in season; and while the gardens are seen at their best in spring and early summer, the bareness of the deciduous trees in the later autumn and winter is relieved by the freshness of the evergreens. At any period of the year one can literally walk into Australian bushland. No description can touch the fringe of the varied beauty of these gardens, one of the greatest joys of the people, gardens which rank with the finest in the world.

**The Treasury Gardens.**—The Treasury Gardens flank the far eastern boundary of the city and are open to three streets, Spring, Flinders and Lansdowne, and are a fitting adjunct to that noble erection, the Treasury Buildings, on the north. They fall steeply away from the terrace-like way called Treasury Place, that runs the full length of the south side of the building. To stand on this terrace and look over the tree tops of great poplars and silver beeches, elms and Moreton Bay figs, beyond which the Yarra valley broadens out, is a beautiful sight. One gets a view towards Government House, the Botanical Gardens and South Yarra hill. The main ornament in this rectangular block is a Japanese garden with its miniature lake and bridges.

**The Fitzroy Gardens.**—The Fitzroy Gardens, covering 64 acres, are a continuation of the Treasury Gardens, from which their western boundary is separated by Lansdowne Street. They are very beautiful and serve well the pedestrian public, the city workers and those who reside in the city and near suburbs. They are probably more used than the other gardens, being a short and pleasant "cut" to and from the city and afford a delightful breathing space. Their beauty lies in the magnificent straight avenues of elms, poplars, limes and planes skirting well-kept lawns. Replicas of classic statuary add here and there to their interest and charm. The distribution of lawns and flower beds is very pleasing. A small Greek temple crowns the north-east point and fits well the dignity of the groves below. A tea kiosk is attractively placed and the conservatory is noted for its fine periodic displays of rare and beautiful flowers. Recently Captain Cook's cottage sent out from England, the centenary gift of Mr. Russell Grimwade, has been erected in these gardens.

**The Zoological Gardens.**—The Zoological Gardens lie due north of the city, and are conveniently reached in a short journey by several routes of tram and train. They cover an area of approximately 45 acres in the domain known as Royal Park. The Zoological Society was founded seventy years ago by Mr. Edward Wilson, the founder and first proprietor of *The Argus*. After a somewhat troublesome early history, largely due to the fact that its original purposes having been identified by the founders with public education, its statute of incorporation forbade the Royal Zoological Society responsible for its administration to charge higher admission fees than sixpence for adults and threepence for children. This left the Society dependent on Governments' subsidies, which were always inadequate for the prescribed purposes of the institution. The zoological collection therefore became necessarily

poorly accommodated, and the gardens fell into a state of disrepair. The Government led by the late Sir William McPherson, realizing this condition, came to the rescue of the Society, the management of which was reconstituted under the leadership of Sir Colin MacKenzie and later Mr. Ambrose Pratt, the present President. The Government then supplied the Society with sufficient funds to provide for the complete reconstruction of the building and a very large extension of the collection, a work that took several years to complete. It was carried out on the soundest scientific lines, with a complete recognition of the necessity to provide the shelter of still air for beasts of the jungle. By 1934 the collection had become one of the most important and varied in Australia, and the animals were housed as well as or better than any similar collection in the world, with the result that the "Zoo's" death rate became the lowest in the world; nor has any case of tuberculosis ever arisen. In 1934 a still more important departure occurred, and the gardens today comprise a special enclosure of four acres which contain, for the first time in the history of the world, a genuinely representative collection of the indigenous fauna of Australia, each type being accommodated as far as possible in its natural condition. To Mr. Ambrose Pratt is due the credit for having been the first to recognize the importance of surrounding jungle animals with still air, with the maximum of sunlight and the minimum of moving air. The Walter and Eliza Hall Institute, which is attached to the Melbourne Hospital, has for some years conducted its researches on snake venom in the Zoological Gardens, which contain the only snake farm in Australia. The collection is representative of the fauna of all countries. One section contains a young platypus which is growing and thriving in a specially designed platypusary, adapted from the original design of Mr. Robert Eadie, of Badger Creek, near Healesville, who has successfully kept his platypus "Splash" in captivity for two years now—a world's record.

**Flagstaff Gardens.**—Flagstaff Gardens, bordered by Latrobe, William and King Streets, belong to the early history of Melbourne. In those days a flagstaff stood on the top of the hill which commanded a view of Port Phillip Bay, and when a ship was sighted a flag was hoisted to announce its arrival. The side of the hill was used as a burial ground.

**Gardens Beyond the City.**—Outside the city the story of the public gardens as here told is continued. It would be safe to say that every suburb within ten miles of the post office has a garden or park, or both, under municipal control. The people realize that playgrounds and breathing spaces, parks and gardens are a vital necessity. And when one travels through South Yarra, Toorak, Malvern, Armadale, Hawthorn, Kew, St. Kilda, Brighton and other suburbs, one realizes the number of fine private gardens, especially in the case of the old established homes and large estates, which have grown to such perfection in the course of many years. Unfortunately, there has been inevitably the "cutting up" of some of these old estates and as a consequence their beauty of parkland and garden has disappeared. But many of the stately homes of Toorak abide.

#### The Churches.

The Anglican Cathedral of St. Paul is situated at the intersection of Flinders and Swanston Streets on the site of the old St. Paul's Church. Its three spires can be seen far off as one approaches the city from the St. Kilda Road. Its architect was the architect of Truro Cathedral in Cornwall. Placed in a busy part of the city, amid ceaseless traffic, one feels the contrast of the peace and quiet within. The visitor will appreciate the singing of the highly trained choristers and the fine organ performances of their teacher, Dr. Floyd. Outside the cathedral is the bronze statuary group of Flinders, the work of the late Web Gilbert.

St. James' Old Cathedral is the oldest church in Melbourne. Its foundation stone was laid in 1839 at the lower end of Little Collins Street, the site being a sheep's pen owned by Batman. It was reerected, stone for stone, in 1913 in King Street near Batman Street, where its beautiful tower overlooks Flagstaff Gardens.

St. Patrick's Cathedral (Roman Catholic) stands on Eastern Hill, a massive blue-stone pile with its spires still uncompleted. A spacious interior, great columns and mellow light from its stained glass distinguish this cathedral, formerly St. Patrick's Church, which was opened in 1859.

Older than it, however, is St. Francis' Church, also Roman Catholic, at the corner of Elizabeth Street and Lonsdale Street, the foundation stone of which was laid in 1841. It is the second oldest church in the city.

St. Peter's Church of England on Eastern Hill, opposite St. Patrick's, dates back to 1841.

The two churches, Scots' and the Independent, which are such a fine feature of Collins Street, have already been mentioned. They have replaced those built in the late thirties of the last century.

Wesley Church in Lonsdale Street has given a special feature to the religious life of Melbourne by the institution of the "Pleasant Sunday Afternoon", when members of any denomination are invited to deliver addresses on subjects of general interest.

The Baptist Church in Collins Street, a little higher up the street than the Town Hall, is one of quiet distinction in its classic exterior.

John Knox's church in Swanston Street, opposite the Public Library, is a beautiful example of Gothic architecture, built in 1847. The church is now owned by the Church of Christ.

#### CONCLUSION.

The foregoing account makes no pretensions or claims to have dealt adequately with the public buildings, the gardens and parks and the many places of interest to a visitor seeing Melbourne for the first time. In a city with more than 200 miles of streets, with a steady expansion of the business and residential areas, the old order is giving way to the new; and following the architectural fashions of the modern world, the erection of more imposing buildings, both public and private, goes hand in hand with the compelling idea of adding to the beauty of the city and its environs. Unfortunately, Melbourne is without its Baedeker, though it has its efficient Tourist Bureau; its excellent transport system of trains and trams makes the quest of things and places worth seeing a pleasing and by no means difficult adventure.

#### ACKNOWLEDGEMENTS.

In preparing this account the writer has consulted:

1. "The Story of Melbourne", by Mr. Alfred S. Kenyon, M.I.E.Aust., M.A.I.M.M., President of the Historical Society of Victoria.

2. "The National War Memorial of Victoria", by Mr. Ambrose Pratt, President of the Zoological Society of Victoria, who has kindly furnished information about the Zoological Gardens.

3. The booklet of the National Art Gallery of Victoria.

### British Medical Association News.

#### SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on November 29, 1934, in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, Dr. A. J. COLLINS, the President, in the chair.

#### Lymphadenoma.

DR. HAROLD RITCHIE read a paper entitled: "Lymphadenoma" (see page 197).

DR. G. F. S. DAVIES read a paper entitled: "Hodgkin's Disease" (see page 199).

Dr. L. Utz thanked the speakers for their interesting papers and for having shown the subject from different angles precisely and fully. He had been very greatly interested in this disease for three or four years and had made it his business when he was abroad eight months previously to see most of the workers on the subject. Three years previously Dr. Utz and Dr. Keatinge had written a paper in which they had tried to discuss the subject from every aspect; they submitted what they thought to be an up-to-date treatise, delving into the literature and trying to offer a survey of the opinion of the world. Just as this paper was published new work was done, and as soon as Dr. Utz left Australia and reached England he saw Gordon, Van Rooyen and others, and was upset to find that the basis of his and Keatinge's work, that of l'Espérance, had been proved incorrect. Without doubt l'Espérance's work did not stand repetition. He had therefore been most interested in seeing the *résumé* of Gordon's work and Van Rooyen's repetition of it, and came to the same conclusion as Gordon, that some virus-like substance was the cause of the disease. Unfortunately Gordon could not reproduce the disease from one animal to another. Then came German and British work. It was found that normal bone marrow contained all the elements to produce some similar condition in rabbits and guinea-pigs. Gordon claimed that his glandular substance (he first thought it was a virus and then a toxin) had a different action from normal bone marrow. He was so certain of this that he and Van Rooyen used the Gordon test (as it was called) as a diagnostic test in this disease. They inoculated rabbits with gland that had been kept on ice and treated in emulsion, and the animal developed encephalitic symptoms. Gordon found that the introduction of glands other than Hodgkin's disease glands did not give this result. All other glands gave negative results. Consequently, after many tests, they found that they could accept the Gordon test as strong evidence of a positive diagnosis of Hodgkin's disease. Dr. Utz said that at Saint Vincent's Hospital, at Edinburgh and at Guy's Hospital many were adopting it as a strong diagnostic test, almost as strong as the histological diagnosis. Naturally Dr. Utz had been very disappointed at the disproof of his work. But at the end his theory was used at Saint Vincent's Hospital in what was called a "novel" type of treatment. On their assumption that the disease was due to an avian tuberculosis, bird serum was used as one type of treatment. When Dr. Utz saw Gordon and Van Rooyen they gave him heart in saying that the patient's response was probably antiviral but still specific. They encouraged him to develop this form of treatment, but with other sera or gland substances. The treatment used at Saint Vincent's Hospital was far from perfect and the results were not meteoric, but he felt that a great deal of good might be done by the routine adoption of this treatment. He had had surprising reports from patients themselves of their progress, the results in many cases lasting for five years. A short time ago Dr. Utz had had a letter from Van Rooyen in which he spoke of another form of treatment. Dr. Utz had referred a patient to Gordon and Van Rooyen from Saint Vincent's Hospital. In this letter, dated September 10, Van Rooyen said that for this patient Gordon suggested deep X rays and sensitized autogenous tissue vaccine. Dr. Utz thought it worth while to progress in some form of treatment such as that outlined. In a letter written in August, 1933, Van Rooyen had said that, in regard to Dr. Utz's own work, the substance used might be simply an antiviral serum, and that he was confident that Dr. Utz's work was therapeutically based on correct lines.

In conclusion, Dr. Utz asked Dr. Davies whether he had ever thought of an explanation for fibrosis in Hodgkin's nodules. At Saint Vincent's Hospital Dr. Utz noticed that glands that seemed to progress most quickly were found at autopsy, when serial sections were made, to be 85% fibrous tissue. How could fibrous tissue grow at this rate?

DR. LEILA KEATINGE said that they had treated forty-five patients suffering from histologically diagnosed lymphadenoma at Saint Vincent's Hospital. All doubtful cases had been excluded. Twenty-four patients were alive, some free from disease, others showing one or two palpable

gland groups, but apparently temporarily inactive. The first patient treated was entirely free from disease five and a half years afterwards, and others four years, three years, and so on. Fifteen patients had died and six others, who they believed were alive, had not reported recently and therefore were not included. Some of these patients had been given serum only, but the majority had, at odd times, been given small doses of X rays to relieve symptoms, for example, dyspnoea. They must accept the fact that lymphadenoma was a generalized disease, because they saw not only lymphatic tissue affected, but also lung parenchyma, bones and spinal cord. Then, if this was so, local removals and local applications of radiation could never hope to cure the disease and were necessarily only palliatives. Therefore some form of general treatment seemed rational. Dr. Utz had told those present how they had arrived at the present routine serum treatment at Saint Vincent's Hospital. They realized that it was imperfect, but nevertheless the following observations on these patients might be of some interest: (i) There was always an improvement in the well-being of the patient; he returned to work and felt better. There might be a psychological factor in this, but patients seemed so definite in their statements that one must note the fact. (ii) One applied a very much smaller dose of X rays than was usually necessary if serum was being given at the same time. (iii) Mediastinal and abdominal glands did not respond to serum in anything like the same degree as did other groups. (iv) The subcutaneous injection of serum to the patient gave a definitely marked local reaction which never occurred in cases of lymphosarcoma, lymphocythemia and allied lymph gland enlargements. This had been very useful before the rabbit encephalitis test came into prominence. (v) The main argument against any definite usefulness of the serum was that in the majority of cases they had had to use X rays as a local and subsidiary treatment when acute symptoms and signs, such as dyspnoea and ascites, had arisen. In the interests of humanity it had been necessary to do this and to refrain from treating the patient purely experimentally. (vi) They had never observed any deleterious effects from serum injections. (vii) All patients, no matter how ill, had shown some definite improvement with serum.

In conclusion, Dr. Keatinge wished to thank Dr. Ritchie and Dr. Davies for their excellent and interesting papers, but at the same time she wished to correct an idea some might have formed from Dr. Ritchie's paper concerning the work of Dr. Utz and herself. They had set out to confirm the work of Elise l'Espérance. They did originally have the idea, as Dr. Utz had explained, that the causative organism was an avian tubercle bacillus, but they did not at any time find tubercular nodules in the hens, which was l'Espérance's strong claim. This fact they had published in THE MEDICAL JOURNAL OF AUSTRALIA, and they now recognized that these claims made by l'Espérance were unfounded.

Dr. A. S. WALKER said that two years ago, when he was in England, he was fortunate in being able to see something of Gordon's work on lymphadenoma. In regard to his test, Gordon adopted extraordinary precautions. The patients tested were submitted first to a careful and independent clinical examination; portion of the gland was subjected to histological examination and portion to biological test. The result of the biological test was given independently of other findings. Dr. Walker had seen rabbits which after such tests presented unique appearances, due to the apparently specific encephalitis. When Dr. Walker saw this work it had not reached its present stage; then Gordon thought that a virus was the cause; now this was doubtful, though it was not disproved. If there was something in the cells that could cause such toxic effects in animals, surely it would be found in conditions other than lymphadenoma, unless it was of a specific nature. It seemed to be the lymphadenoma substance that produced this curious characteristic encephalitis, whether it was a viral toxin or not. This hypothesis seemed to present a very promising line of research. Positive results were obtained in apparently 100% of cases, Gordon had referred to the work of Utz and Keatinge and thought that their results were obtained

through some antiviral substance. Dr. Keatinge had pointed out that the patients in their care had not received one form of treatment only. Lymphadenoma was a disease in which remissions occurred when no treatment whatever was being given, and also an extraordinary increase of symptoms might occur for no apparent reason. For this reason results were difficult to assess from the clinical point of view, but it was well worth while to continue with the work. Much attention had been given to viruses in recent years. Collier had recently referred to the "encaged virus" in the nervous system in lethargic encephalitis and he advanced the idea that this condition, with all its sequelae, was due to one persistent virus in the nervous system and that the virus might remain latent for a long time. Parallels might be suggested in tuberculosis and syphilis. Therefore it was not impossible that a disease that might last so long as lymphadenoma might be due to a living virus.

In conclusion, Dr. Walker congratulated Dr. Ritchie on his clear clinical picture of the condition, and Dr. Davies on his excellent demonstration of the histology.

Dr. EVA SHIPTON said that there were one or two things that she wanted to ask Dr. Davies. In regard to the eosinophile cells in this condition, she believed that it had been established by Lang's work on extramedullary haematopoiesis that the eosinophile cells were products indirectly of the reticular cells, and that the reticular cells also gave rise to the fibrous tissue; and although he did not refer specifically to Hodgkin's disease, in this condition there was simply an alteration of the stimulus by which any type of cell could be made to develop. This explained the formation of the megacaryocytic type of cell, which could also be traced back to reticular cells. These reticular cells were universal throughout the body, and this explained why Hodgkin's disease was a disease not confined to a single organ, but was a disease of any organ. As to the blood count, in this connexion it was a pity that the hematologist did not always interpret the blood count for clinicians, who sometimes missed important points.

Dr. FIASCHI asked Dr. Ritchie if he knew what Nicholas Favre's disease was.

Dr. KEITH INGLIS spoke of the importance of sending along a proper piece of material to the histopathologist in this very difficult disease. Some people had an idea that if a small gland was sent along it would be easier to recognize the disease in its early stages, and that a large gland was likely to be fibrotic. Dr. Inglis, however, preferred a large one. Sometimes he had to say that he could not express an opinion on such a small specimen; but if the specimen had been larger he would have been able to do so. Diagnosis was not so easy as might be thought from the excellent slides of Dr. Davies. In some instances the interpretation was very difficult.

In conclusion, Dr. Inglis spoke of the difficulty of distinguishing between reticulum-celled lymphosarcoma and Hodgkin's disease.

Dr. C. G. WILLCOCKS asked Dr. Ritchie concerning the details of treatment with deep X rays, and he asked Dr. Davies what happened to the glands when they actually went down with deep X ray treatment. He wondered where the stuff actually went to, and he did not mean to be entirely humorous. He wondered whether glands had been examined after they had been subjected to deep X rays. There were patients with glands that came up in a night, and a small dose of deep X rays would make them go down almost at once. Dr. Willcocks wondered why this was, what enlarged the glands, and whether the swelling was actually due to oedema.

Dr. A. W. HOLMES A COURT said that the chief point brought out was the difficulty in diagnosis; the difficulty was not only a clinical one, since it was often not removed when biopsy was carried out. Yet some clinical features made recognition of the condition possible even without biopsy. In a group of glands there was a variability of consistency of the gland masses which was a peculiarity of this condition, probably due to the varying amount of interstitial fibrous tissue. Then there was a type which exhibited the Bell-Ebstein phenomena in Hodgkin's disease.

Dr. Holmes & Court mentioned the case of a boy who had been in hospital with a temperature of 105° F. and had been febrile for a fortnight; he then had an apyrexial period. This sequence had been reproduced on several occasions. Hearing the symptoms and without seeing the patient, Dr. Holmes & Court had suggested a diagnosis of lymphadenoma, which subsequently proved to be correct.

Dr. KENNETH SMITH supported Dr. Ritchie in a plea that large teaching hospitals should endeavour to obtain good statistics. Statistics in their present condition were not of much value in the investigation of disease. Good statistics cost money, but he thought that expenditure of money on records departments would be well worth while.

Dr. A. J. COLLINS, on behalf of the New South Wales Branch of the British Medical Association, thanked the readers of the papers and also those who had taken part in the excellent discussion.

Dr. Ritchie, in reply, thanked members for the kind way in which they had received his remarks. He wished especially to congratulate Dr. Davies on his excellent illustrative description of the pathology of the disease.

In reply to Dr. Fiaschi, Dr. Ritchie said that Nicolas Favre's disease was really *lympho-granuloma venereum*; a venereal disease. It commonly involved glands in the inguinal region in the male and gave rise to suppurative changes. In women it involved the lymphatics draining the cervix uteri and often gave rise to stricture of the rectum from a secondary cellulitis. The virus of this disease had been isolated by Frei. *Lympho-granuloma venereum* was also a variety of viral disease in glandular tissue, benign in character, but not related to lymphadenoma. There was a blood test for *lympho-granuloma venereum*.

Dr. Willcocks asked about X ray therapy; Dr. Ritchie had said nothing about therapy in his paper. Dr. Ritchie replied that deep X ray therapy certainly did good, but its effect was variable, and he had never seen a case in which permanent benefit was gained, though the primary response was sometimes magical, especially with mediastinal glands and in the relief of dyspnoea. But lymphadenoma was an affection of reticular tissue and they could not hope to cure a generalized disorder by the application of X rays to various groups of glands.

Dr. Ritchie was glad that Dr. Kenneth Smith sided with him about the keeping of good records. It was very important and they should try to see that hospital records were better kept. Before a junior house physician was allowed to append his diagnosis he should be overseen by some senior, preferably his honorary physician.

Dr. Davies, in reply, thanked members for the way in which they had appreciated his slides. Dr. Uts had asked about fibrosis of lymph glands. Dr. Davies had noticed that the degree of fibrosis might vary greatly in different glands from the same case. In the mediastinal glands from one case the fibrous tissue was dense and hyaline. In one from high up in the neck, near the base of the skull, the fibrosis was advanced, but the hyaline change was not so pronounced, while a gland from the groin showed only slight fibrosis, and in this the lymphadenomatous changes were comparatively early.

In regard to what Dr. Shipton had said about eosinophile cells, Dr. Davies thought also that they developed from reticular cells.

In reply to Dr. Willcocks, Dr. Davies said that many glands that showed necrosis had had deep X ray treatment, yet some others which had had no radiation therapy contained necrotic areas. The case already referred to, in which some glands were very fibrotic and others showed early changes, had been treated by deep X rays. Where one set of glands was becoming fibrotic the disease was starting in another group.

#### NOMINATIONS AND ELECTIONS.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Johnson, David William, M.B., B.S., 1933 (Univ. Sydney), Sydney Hospital, Sydney.

Watt, John Langston, M.B., B.S., 1933 (Univ. Sydney), Tenterfield.

Crisp, Arthur Charles, M.B., B.S., 1933 (Univ. Sydney), Nepean Dam, Bargo.

### Correspondence.

#### THE WHITE MAN IN THE TROPICS.

SIR: The address given by Dr. Grenfell Price on the white man in the tropics opens up many interesting problems. I wish, however, to limit my observations to ascertained facts. From the Queensland border to the Indian Ocean, north of the tropic of Capricorn, there are 6,400 white people in this enormous territory and with them the general and infantile mortality rates are high, which is not remarkable when the conditions of life both social and physical are realized.

About 4,500 people live in our Northern Territory on which £17,000,000 have been spent. But the country, owing to its climate and soil, does not invite settlement.

I am informed that on the south coast of Java there is country similar in character which even the 40,000,000 inhabitants of Java do not or cannot colonize.

But the statistical evidence recently made available by the Commonwealth Statistician, Mr. McPhee, tells another tale for tropical Queensland. The population of tropical Queensland has risen in the ten years 1923 to 1932 at the rate of 2% per annum. The remainder of Australia has risen at the rate of 1.5%. The birth rate in tropical Australia in 1932 was 21.25 and for the rest of Australia 16.81. The death rate in tropical Australia in 1932 was 9.41 and in tropical Queensland 9.07 and in non-tropical Australia 9.07 in the cities, but 8.65 as a whole. The infantile death rate in 1932 was 42.47 in the tropics and in non-tropical Australia 42.16, but the metropolitan rate was 44.35 and the extra-metropolitan rate 39.74. The population of tropical Queensland is 170,000.

Here then is a rapidly increasing healthy population. Obviously the difference in settlement of the several parts of tropical Australia is due to economic causes. People can find occupation and profit in tropical Queensland but not easily in Northern and north-west Australia. The pastoral and mining industries may find a home in these empty territories, especially if means of communication are improved, and the Council of Scientific and Industrial Research has the problem in hand. It is desired to find a breed of animals which suits the country.

Tropical Queensland is the only tropical country in the world so far practically free from tropical disease. If it possessed an infected indigenous population there is little doubt of the disastrous result which would ensue.

Dr. Price's reference to white women in tropical Australia recalls the fact that I obtained, through the courtesy of Mr. Amery, on two occasions plans and specifications of the houses designed and built in the Panama Canal zone, and forwarded one set of them to the Commonwealth Government. I do not know whether these designs have ever been adopted in tropical Australia, but I have seen many unsuitable houses in that country.

The conclusion reached is that white people do live and thrive in most parts of tropical Australia where it is possible to conduct a profitable industry. The failure in other parts is not climatic, but economic. No one, however, can examine the problem without cordially agreeing with Dr. Price that a successful utilization of tropical Australia is likely to succeed only when it is taken out of political and placed in scientific hands. Recently we witnessed the spectacle of an attack made by politicians on those who

told the truth, namely, that the Northern Territory is poor to a degree. Something may be done with it, but only by facing unpalatable facts.

Yours, etc.,

JAMES W. BARRETT.

103-105 Collins Street,  
Melbourne,  
January 30, 1935.

### Obituary.

WILLIAM HENRY BASIL O'NEILL.

We regret to announce the death of Dr. William Henry Basil O'Neill, which occurred on February 5, 1935, at Neutral Bay, New South Wales.

### Books Received.

AIDS TO PSYCHIATRY, by W. S. Dawson, M.A., M.D., F.R.C.P., D.P.M.; Third Edition; 1934. London: Baillière, Tindall and Cox. Pp. 325. Price: 4s. 6d. net.

AIDS TO THE ANALYSIS OF FOOD AND DRUGS, by C. G. Moor, M.A., F.I.C., and W. Partridge, F.I.C.; Fifth Edition, revised and partly rewritten by J. R. Nicholls, B.Sc., F.I.C.; 1934. London: Baillière, Tindall and Cox. Pp. 330. Price: 5s. net.

### Diary for the Month.

- FEB. 19.—New South Wales Branch, B.M.A.: Ethics Committee.  
FEB. 19.—Tasmanian Branch, B.M.A.: Council.  
FEB. 22.—Queensland Branch, B.M.A.: Council.  
FEB. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.  
FEB. 27.—Victorian Branch, B.M.A.: Council.  
FEB. 28.—South Australian Branch, B.M.A.: Branch.  
MAR. 1.—Queensland Branch, B.M.A.: Branch.  
MAR. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.  
MAR. 5.—Tasmanian Branch, B.M.A.: Council.  
MAR. 6.—Western Australian Branch, B.M.A.: Council.  
MAR. 6.—Victorian Branch, B.M.A.: Branch.  
MAR. 7.—South Australian Branch, B.M.A.: Council.  
MAR. 8.—Queensland Branch, B.M.A.: Council.  
MAR. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee; Ethics Committee.  
MAR. 19.—Tasmanian Branch, B.M.A.: Council.  
MAR. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.

### Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser", pages xiv to xvii.

- CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Honorary Officers.  
CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officer.  
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.  
PUBLIC SERVICE BOARD, SYDNEY, NEW SOUTH WALES: Junior Medical Officer.  
ROYAL HOSPITAL FOR WOMEN, PADDINGTON, NEW SOUTH WALES: Honorary Physician, Resident Medical Officer.  
ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.  
SAINT VINCENT'S HOSPITAL, MELBOURNE, VICTORIA: Honorary Officers.  
THE BENEVOLENT SOCIETY OF NEW SOUTH WALES: Honorary Officers.  
THE GOODDOGA DISTRICT HOSPITAL, GOODDOGA, NEW SOUTH WALES: Medical Officer.  
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

### Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Peteraham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phoenix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 267, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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